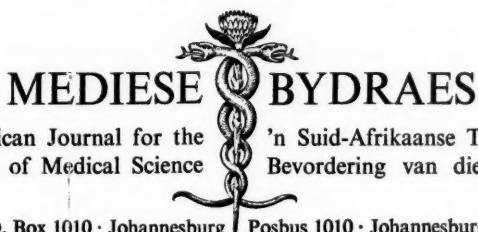


MEDICAL PROCEEDINGS



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EDITORIAL · REDAKSIONEEL

THE STRESS ELECTROCARDIOGRAM

Elsewhere in this issue Dr. B. A. Bradlow and Dr. M. M. Zion, of Johannesburg, publish a comprehensive study of the influence of exercise, under controlled conditions, on the ECG pattern. The prognostic significance of the effect of exercise on the electrical changes in the heart is obvious for ordinary clinical and diagnostic practice. It is also clearly important in insurance medicine.

Largely as a result of the stimulus provided by the statistical studies of Robb, Marks and Mattingly,¹ of the Metropolitan Life Insurance Company of New York, this application of the effort electrocardiogram has come to the fore increasingly in recent years. As Gubner² has recently remarked:

'Indeed, the electrocardiographic response to stress in many ways bears more closely on the problems of coronary insufficiency and angina pectoris than do morphologic changes in the coronary arteries, such as may be elegantly visualized by coronary arteriography. It is well recognized that no necessary, or at times even close correlation obtains between organic changes in the coronary arteries and myocardial involvement or between these and electrocardiographic changes and anginal pain.'

To-day an ECG investigation of a middle-aged subject for insurance purposes must be regarded as quite incomplete if a stress test has not been done.

DIE BELASTINGSELEKTROKARDIO- GRAM

Elders in hierdie uitgawe publiseer ons 'n omvattende referaat deur dr. B. A. Bradlow en dr. M. M. Zion, van Johannesburg, oor die invloed van oefeninge, onder gekontroleerde toestande, op die EKG-patroon. Die prognostiese betekenis van die effek van oefening op elektriese veranderinge in die hart lê voor die hand in die gewone kliniese en diagnostiese praktyk. Wat versekeringsgeneeskunde betref, is dit heel duidelik ook van belang.

Grotendeels as gevolg van die aansporing voortvloeiende uit die statistiese studies van Robb, Marks en Mattingly,¹ van die Metropolitan Life Insurance Company van New York, het hierdie toepassing van die belastingselektrokardiogram gedurende die afgelope jare in 'n steeds groter mate op die voorgrond getree. Soos Gubner² onlangs gesê het:

'Inderdaad, die elektrokardiografiese reaksie op belasting staan in menige opsig in nouer verband met die probleme van koronêre ontoereikendheid en angina pectoris as die morfologiese veranderinge in die koronêre slagare, soos dié wat elegant deur middel van koronêre arteriografie besigtig kan word. Allerweë word daar erken dat daar geen noodwendige of, by wyle, selfs noue verwantskap bestaan tussen organiese veranderinge in die koronêre slagare en hartspierverwikkeling, of tussen

1. Robb, G. P., Marks, H. H. and Mattingly, T. W. (1957): Trans. Assoc. Life Insur. Med. Dir. Amer., **40**, 52.
2. Gubner, R. (1961): J. Occup. Med., **3**, 110.

1. Robb, G. P., Marks, H. H. en Mattingly, T. W. (1957): Trans. Assoc. Life Insur. Med. Dir. Amer., **40**, 52.
2. Gubner, R. (1961): J. Occup. Med., **3**, 110.

The adequately performed stress ECG (as defined by the authors on the basis of the work described by Wood)³ provides a delicate indicator which may unmask underlying myocardial ischaemia, particularly when this is asymptomatic. In this respect the Wood effort test has considerable advantages over the Master 2-step test.⁴ A properly conducted stress test is therefore an important part of the investigation of the efficiency and the potentialities of the heart. Bradlow and Zion recommend it as 'part of a routine check in any person over the age of 35 and under the age of 60.' After the age of 60 the incidence of a diminished cardiac reserve due to myocardial ischaemia is so considerable that a large number of positive results in the stress test will be obtained merely as the inevitable accompaniment of the ageing process, without any significance in respect of an appreciable reduction in life expectancy.

An important new finding that emerges is the observation by Drs. Bradlow and Zion that, after exercise, sinus arrhythmia (without accompanying chest pain) may emerge in association with depression of the ST-segment. The authors attach prognostic significance to this change after effort as indicating a confirmatory sign of myocardial ischaemia.

Experienced cardiologists have probably also come across the situation in which the exercise test induces angina, without depression of the ST-segment, but with a sinus arrhythmia. In the present state of our knowledge this syndrome may possibly be regarded as an expression of myocardial ischaemia, despite the absence of depression of the ST-segment.

The survey published by Bradlow and Zion emphasizes the extremely important and necessary role played by the electrocardiogram in the practice of modern cardiology. The stress ECG forms an integral part of a medical insurance examination and its omission could to-day be regarded as a serious inadequacy in the examination of a proposer.

SMITH, KLINE AND FRENCH LABORATORIES AWARD FOR POST-GRADUATE CLINICAL STUDY IN SOUTH AFRICA

1961 FELLOWSHIP

This award has been established by a grant from SKF Laboratories (Pty.) Limited, P.O. Box 784, Port Elizabeth. This is the South

laasgenoemde en elektrokardiografiese veranderings en angina-pyn nie.

'n EKG-ondersoek van 'n middeljarige pasiënt vir versekeringsdoeleindes moet vandag as volkome onvolledig beskou word as 'n belastingstoets nie uitgevoer is nie.

Die doeltreffend uitgevoerde belastings-EKG (soos gedefinieer deur die skrywers op grondslag van die werk dat deur Wood³ beskryf is) verskaf 'n delikate aanwyser wat enige onderliggende hartspierisemie kan ontmasker, veral as dit asimptomaties is. Wat dit betref, is Wood se belastingstoets aansienlik beter as die Master-2-staptoets.⁴ 'n Behoorlik uitgevoerde belastingstoets vorm derhalwe 'n belangrike deel van enige ondersoek na die doeltreffendheid en die potensiaaliteit van die hart. Bradlow en Zion beveel dit aan as 'deel van die roetine-ondersoek van enige persoon bo die ouderdom van 35 en jonger as 60'. Na die ouderdom van 60 is die voorkoms van 'n verminderde hartreserwe volgende op hartspierisemie so groot dat 'n aansienlike aantal positiewe resultate met die belastingstoets verkry sal word bloot as die onvermydelike metgesel van die verouderingsproses, en sonder enige besondere betekenis wat betref 'n waarneembare vermindering van die lewensverwagting.

'n Belangrike nuwe bevinding wat aan die lig gekom het, is die waarneming deur drs. Bradlow en Zion dat, ná oefening, sinus-aritmie (sonder bygaande borspyn) te voorskyn kan tree in assosiasie met depressie van die ST-segment. Die skrywers heg prognostiese betekenis aan hierdie verandering na belasting, en meen dat dit as bevestiging van hartspierisemie dien.

Ervare kardioloë het waarskynlik ook reeds te kampe gehad met toestande waar die oefeningstoets angina te voorskyn roep sonder depressie van die ST-segment, maar met 'n sinus-aritmie. Vir sover ons kennis op die oomblik strek, kan hierdie sindroom miskien beskou word as 'n openbaring van hartspierisemie, ondanks die feit dat daar geen depressie van die ST-segment is nie.

Die opname van Bradlow en Zion beklemtoon die buitengewoon belangrike en noodsaaklike rol wat die elektrokardiogram in die moderne kardiologiese praktyk speel. Die belastings-EKG is 'n integrerende deel van mediese ondersoek vir versekeringsdoeleindes, en die weglating daarvan moet vandag beskou word as 'n ernstige gebrek in die ondersoek van die persoon wat aansoek om versekering doen.

SMITH, KLINE EN FRENCH LABORATORIES SE BEURS VIR NA-GRAADSE KLINIESE STUDIE IN SUID-AFRIKA

1961 SE TOEKENNING

Hierdie beurs is moontlik gemaak deur 'n toelae wat deur SKF Laboratories (Pty.) Limited, Posbus 784, Port Elizabeth, beskikbaar gestel

3. Wood, P., McGregor, M., Magidson, O. and Whittaker, W. (1950): Brit. Heart J., **12**, 363.
4. Master, A. M., Friedman, R. and Dack, S. (1942): Amer. Heart J., **24**, 777.

3. Wood, P., McGregor, M., Magidson, O. en Whittaker, W. (1950): Brit. Heart J., **12**, 363.
4. Master, A. M., Friedman, R. en Dack, S. (1942): Amer. Heart J., **24**, 777.

African branch of Smith, Kline and French Laboratories Ltd., London.

The Selection Committee (an entirely independent board of medical practitioners) consists of the following:

Prof. J. F. Brock (*Cape Town*);
 Prof. E. H. Cluver (*Johannesburg*);
 Prof. G. A. Elliott (*Johannesburg*);
 Prof. J. H. Louw (*Cape Town*);
 Dr. H. A. Shapiro (*Honorary Chairman, Johannesburg*);
 Dr. M. Shapiro (*Johannesburg*);
 Dr. M. M. Suzman (*Johannesburg*);
 Prof. H. W. Snyman (*Pretoria*).

Applications are invited from registered general practitioners who have been in active practice in South Africa for at least 7 years.

The Bursary is intended for post-graduate clinical study and not for medical research. It is available for not less than a 2-month period at any Medical School in South Africa.

The total value of the Bursary is R600.

The candidate must submit a brief statement of his proposed course of study and indicate the institution at which he intends to undertake it.

No payments will be disbursed to the successful applicant until he has satisfied the Selection Committee that he has been accepted for the period of post-graduate study at a South African Medical School.

Applications must be made on the prescribed form which is obtainable from:

Dr. H. A. Shapiro (*Honorary Chairman*),
 Selection Committee,
 SKF Laboratories Award for Post-Graduate Clinical Study,
 P.O. Box 1010, Johannesburg.

Closing Date for Applications: 30 June, 1961.

is. Die genoemde firma is die Suid-Afrikaanse tak van Smith, Kline and French Laboratories Ltd., London.

Die Keurkomitee ('n volkome onafhanklike raad van mediese praktisyne) bestaan uit die volgende:

Prof. J. F. Brock (*Kaapstad*);
 Prof. E. H. Cluver (*Johannesburg*);
 Prof. G. A. Elliott (*Johannesburg*);
 Prof. J. H. Louw (*Kaapstad*);
 Dr. H. A. Shapiro (*Ere-Voorsitter, Johannesburg*);
 Dr. M. Shapiro (*Johannesburg*);
 Dr. M. M. Suzman (*Johannesburg*);
 Prof. H. W. Snyman (*Pretoria*).

Aansoeke word ingewag van geregistreerde algemene praktisyne wat ten minste 7 jaar lank aktief in Suid-Afrika gepraktiseer het.

Die Beurs is bedoel vir na-graadse kliniese studie en nie vir mediese navorsing nie. Dit is beskikbaar vir 'n tydperk van ten minste 2 maande aan enige Mediese Skool in Suid-Afrika.

Die totale waarde van die Beurs is R600.

Die kandidaat moet 'n kort uiteensetting van sy voorgestelde studiekursus verstrek, en hy moet aandui by watter inrigting hy hierdie kursus wil loop.

Geen geld sal aan die suksesvolle aansoeker uitbetaal word nie totdat hy die Keurkomitee tevrede gestel het dat hy aangeneem is vir die tydperk van na-graadse studie aan 'n Suid-Afrikaanse Mediese Skool.

Aansoek moet gedoen word op die voorgeskrewe vorm wat verkrygbaar is van:

Dr. H. A. Shapiro (*Ere-Voorsitter*),
 Keurkomitee,
 SKF Laboratories se Beurs vir Na-Graadse Kliniese Studie,
 Posbus 1010, Johannesburg.

Die sluitingsdatum vir aansoeke is 30 Junie 1961.

ABSTRACT

THE PATHOGENESIS OF SERUM SICKNESS

The pathogenesis of serum sickness was investigated by Korowajew in 133 diphtheria patients, 80% of whom showed clinical manifestations of serum sickness, mostly of a mild or moderately severe nature. About two-thirds of the patients were children aged 3-15 years. The author distinguishes between four forms of serum sickness:

In the autonomic nervous form of serum sickness there is an increase in parasympathetic nervous tone, without any substantial reaction on the part of the mesenchyma, blood, or lymph; it is more frequently observed in moderately severe cases of the disease and runs a tedious course. The endothelial barrier is apparently only incomplete. The mesenchymal form—involving a rise in the number of lymphohistiocytic elements in the fluid of the cantharides blister—is often encountered during mild serum sickness of short duration; here, the endothelium forms

a barrier which protects the important internal organs from toxic products of the antigen-antibody reaction. Both forms together are met with primarily in severe cases. The serological form, involving a pronounced reaction on the part of the blood without other manifestations, occurs to all intents and purposes only in cases of latent serum sickness. The anergic form is characterized by mild clinical symptoms without any other changes; like the mesenchymal form, it affects chiefly small children. The autonomic nervous form, on the other hand, is particularly common in the 10-15 age group. The explanation for this is that the autonomic nervous system and the mesenchyma are not developed to the same degree in the various age groups and their reactivity is different.

[Korowajew, E. N. (1959): Allergie u. Asthma, 5, 1].

THE EFFORT ELECTROCARDIOGRAM

BERTRAM A. BRADLOW, M.D. (RAND.), M.R.C.P., M.R.C.P.E.*

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and

MONTY M. ZION, M.D. (RAND.), M.R.C.P.

Johannesburg Hospital, Tara Hospital and the University of the Witwatersrand, Johannesburg

Everything has been said already but as no one listens we must always begin again.—*André Gide.*

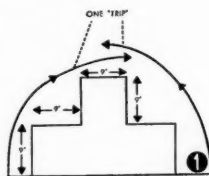
Stress tests invoking the ECG have been used for more than 20 years to obtain objective evidence about the adequacy of the coronary circulation. There is, however, still a great deal of disagreement about their value, due largely to the different criteria adopted by various authors for the interpretation of these tests.

The indications for an effort ECG may be summarized as follows:

1. Equivocal history with a normal resting ECG.
2. An atypical history and an abnormal resting ECG showing, e.g. bundle branch block or minor non-specific T wave flattenings or inversions.
3. When chest pain arises for the first time and acute myocardial ischaemia is suspected but cannot be so diagnosed. In such a case the test has been thought by some to be dangerous. We personally wait for 10 days after such an event before performing the test.
4. As a routine for large insurance policies or pension schemes. It is notorious that applicants for insurance will often minimize or omit symptoms of coronary disease in their history.
5. As a part of a routine check in any person over the age of 35 years and under the age of 60 years.
6. In otherwise unexplained arrhythmias, e.g. atrial fibrillation.

The method of performing the test varies.^{1-3,5} Master *et al.*⁵ uses Tables based on age, sex and weight, and the subject makes a given number of trips or ascents in exactly $1\frac{1}{2}$ or 3 minutes. One trip in this '2-step' test consist of climbing the two 9-inch steps and then descending the other side (Fig. 1). We prefer to exercise the patient on the 2-step until he is pulled up by pain, dyspnoea or fatigue,^{1,4} as fit or athletic people can perform ordinary exercise with such ease that their coronary reserve is not actually tested. We would like to stress that the test should be

performed in a basal state or as near basal as possible. This means that the patient should be fasting or come in about 2 hours after a meal, as the post-prandial ECG may sometimes be abnormal.



The electrodes are left strapped in position during the test and the cable may be left attached while the patient holds it. We make recordings immediately and at 2 or 3 minutes and at 5 minutes after effort and, if necessary, at 2-minute intervals until the ECG returns to normal.⁵ It is probably ideal to record all the leads in the reverse order, starting at V₆, but to make the test less unwieldy, more practical and at the same time very nearly all-embracing, we repeat V₆, V₅ and V₄ in that order to show changes over the left ventricle; V₂, which in the rare case shows changes over the right ventricle, and S₂ as an all-purpose lead. Recently we have felt that aVF yields no useful information and have abandoned this lead. However, we are aware that by restricting ourselves to 5 leads we may be missing the rare positive reactors who would show up in other leads.^{2,5,6} In fact, we have seen cases where V₅ showed only junctional S-T depression, whereas V₆ or less often V₃ or aVF showed ischaemic depression. The changes due to ischaemia may occur at once or be delayed for 2-5 minutes or longer.¹⁴ True isolated inversion of the T wave, when it occurs, usually develops during the late recovery phase.^{3,4,7}

Normally after effort the P wave is elevated in leads 2 and 3, and depressed in lead 1 indicating that the P axis deviates to the right.

* Consultant Medical Officer: Swiss South-African Reinsurance Co. Ltd., and the Northern Assurance Co. Ltd.

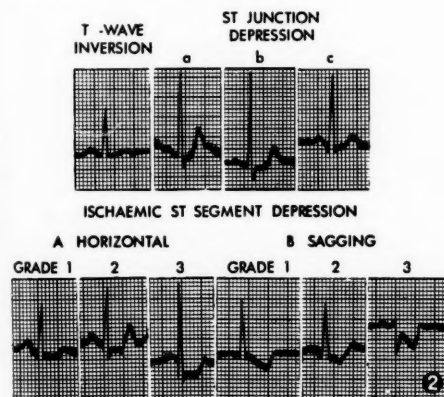
This paper was presented at the Second Congress of the South African Cardiac Society in Cape Town in October 1960.

Thus an inverted P3 may become upright after effort. Duration of the P wave may be slightly increased.⁸

The P-R segment shows slight depression not exceeding 0.15 MV due to accentuation of a normally negative P-Ta segment.⁸ The P-R interval is almost always decreased out of proportion to the increase of the heart rate. Several minutes after effort, when the heart rate slows, the P-R interval may become longer than it is at rest, but not abnormally long.

The QRS duration is usually unchanged but the voltage of QRS is depressed in all leads immediately after effort. It becomes slightly elevated during the following few minutes. There is again a tendency to right axis deviation.

The S-T junction may be depressed due to accentuation of the P-Ta segment, the S-T being a direct continuation of the depressed P-R segment. The ascending limb of the negative P-Ta segment causes the S-T to slope upward so that the angle between S-T and T becomes more obtuse. However, less commonly true depression of the ST junction compared to the P-R segment may occur but the S-T segment still slopes upwards.



The T wave shows primary depression immediately after effort, but becomes elevated one minute later and after 3-5 minutes shows secondary depression. In some normal persons after strenuous effort the T wave may become inverted in leads 2 and 3 and the precordial leads, without any S-T depression. There is a tendency for the T axis to show right deviation and an inverted T3 may become positive.

Immediately after exercise the Q-T duration is slightly decreased but may show a secondary increase later.

The U wave is usually elevated, but if the exercise is strenuous the U wave may disappear.

The abnormal response to effort concerns itself mainly with the S-T segment and less so with the T and U waves.

The P-R segment is usually taken as the base line.^{5,7} Alternatively, a line drawn between the beginning and the end of the P wave, when there is little or no P-R segment, will probably do just as well. The shape of the S-T segment is more important than the degree of depression. Fig. 2 illustrates the different types of depression. Firstly, it shows isolated T wave inversion which, as already pointed out, is a normal phenomenon. The S-T segment is not depressed. S-T junction depression of any degree may be of 3 types:

- Transitory, during tachycardia.
- Prolonged, occurring when the heart has slowed.
- Near-ischaemic. S-T segment straight and nearly horizontal but really, as with the other 2 types, sloping upward.¹⁵ This is the type which resembles the ischaemic depression and may, in fact, hide a few ischaemic depressions. True junctional depression is also a normal phenomenon.

Ischaemic S-T segment depression consists of 2 types. Each type has 3 grades:

Type A shows a horizontal segment without significant T wave changes. Grade 1 is depressed from 0.0-0.9 mm.; grade 2 is depressed from 1.0-1.9 mm., and grade 3 is depressed 2 mm. or more.

Type B has a sagging S-T segment with a biphasic or inverted T wave. It is graded in the same way as is the horizontal segment.¹⁵

It will be noted that the ischaemic S-T depression is identical with that seen in naturally occurring attacks of angina if the ECG is recorded during the attack.

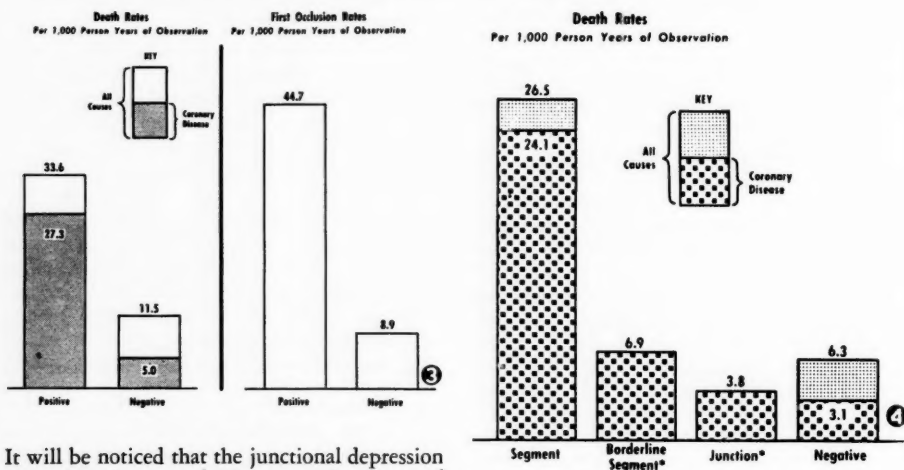
TABLE 1: DEATH RATE FROM CORONARY DISEASE PER 1,000 PATIENT-YEARS OF OBSERVATION

| | |
|---------------------------------|----------------------------|
| Negative—3.1 | ST Junction Depression—2.8 |
| Ischaemic—23.8 | |
| Total | |
| Grade 1—12.9 | Grade 2—21.2 |
| Grade 3—95.1 | |
| (G. Robb & H. H. Marks—Ref. 15) | |

Table 1 shows the death rate from coronary disease per 1,000 patient-years of observation according to the various grades. The coronary death rate¹⁵ in grade 3 is more than 7 times the rate for grade 1.

Fig. 3 shows graphically the death rates and first occlusion rates for positive and negative reactors. Fig. 4 shows the death rates broken up according to the type of S-T depression.

sion is not a sign of ischaemia. A change from negativity to positivity of a T wave is also of no significance. Pure T wave changes are relatively uncommon.



It will be noticed that the junctional depression group has the same death rate as the group of negative reactors. The 'borderline' or 'near-ischaemic' type has a higher death rate, probably because it includes some positive reactors.

The isolated T wave inversions unaccompanied by S-T depression are included among the negative reactors. Isolated T wave inver-

sion is not a sign of ischaemia. A change from negativity to positivity of a T wave is also of no significance. Pure T wave changes are relatively uncommon.

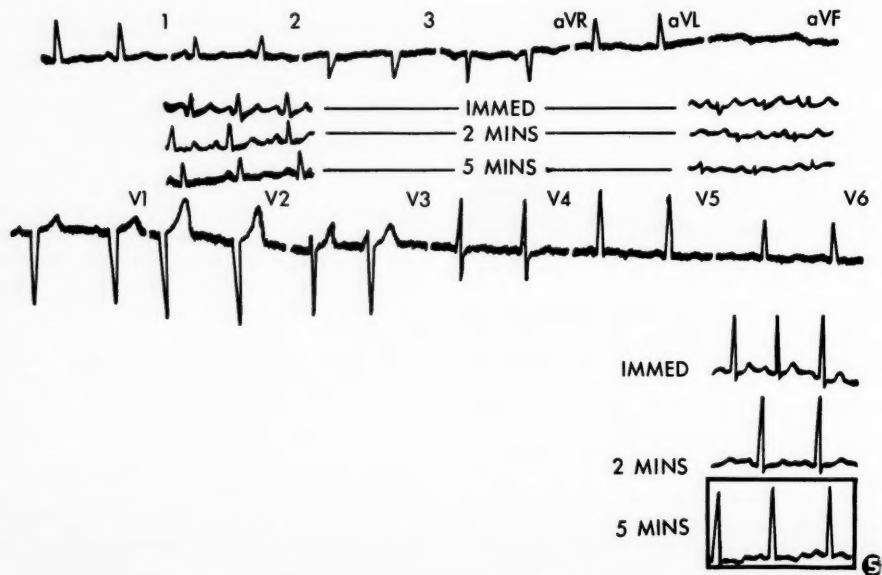


Fig. 6 shows primary depression of T in aVF in the immediate tracing of a 25-year-old man without symptoms.

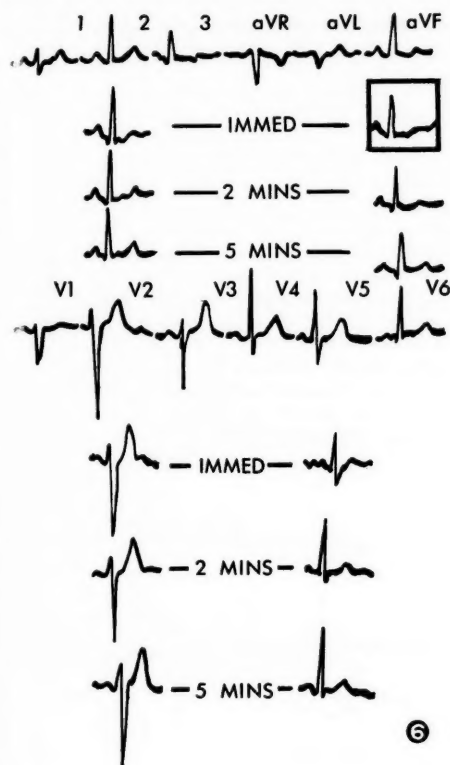


Fig. 8 shows junctional depression of the 'near-ischaemic' type (type C), most noticeable at 2 and at 5 minutes in V5 in a 48-year-old woman with cardiac anxiety and left intra-mammary pain. Notice that the S-T segment still slopes upward.

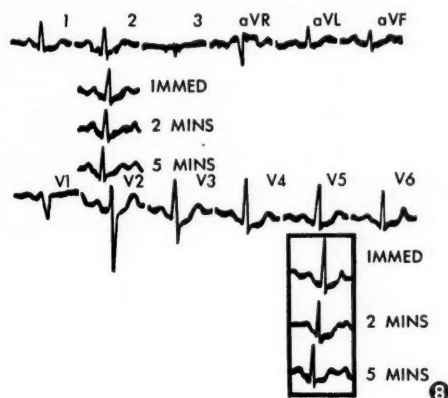


Fig. 9 demonstrates doubtful ischaemia shown by shape rather than degree of depression in V5 at 2 minutes in a 52-year-old woman with a cardiac neurosis.

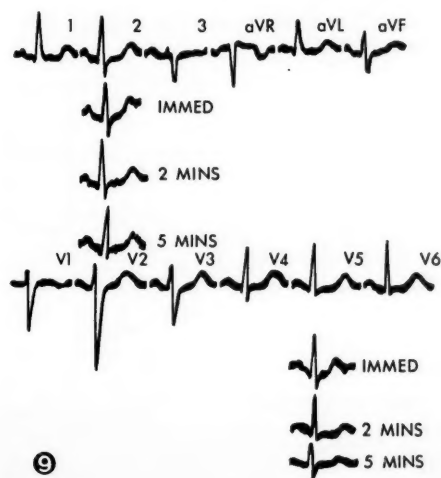
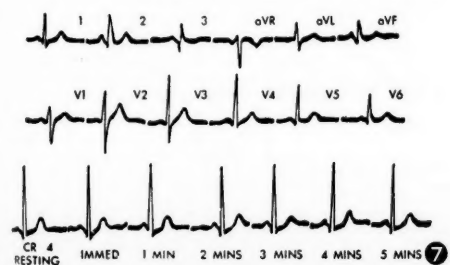
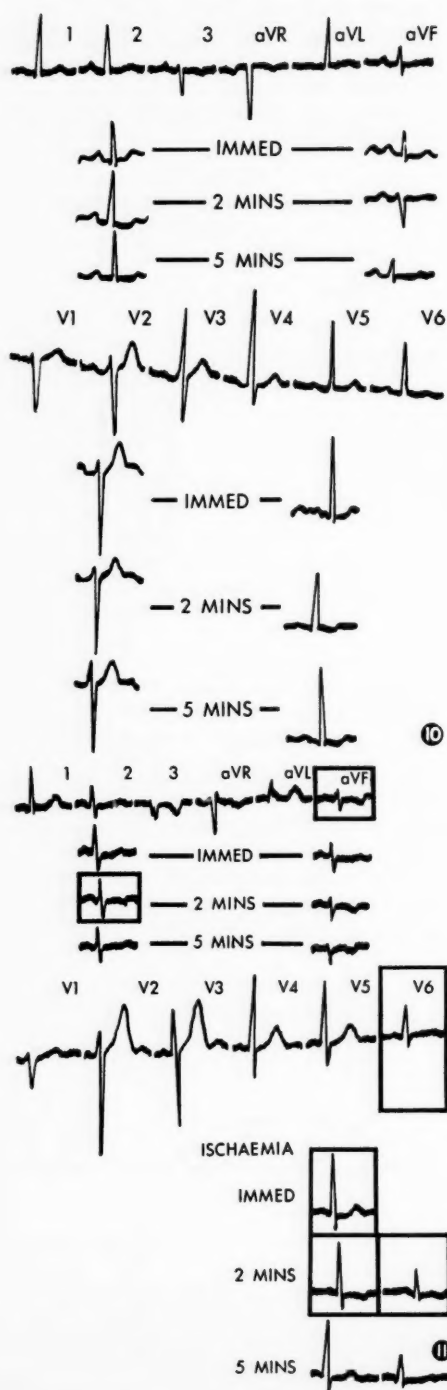


Fig. 7 shows transient junctional S-T depression in a 29-year-old man with a story of severe sharp substernal pain of 2 minutes' duration.





Grade 1 sagging S-T depression in V5 and S2 is shown in a 44-year-old man (Fig. 10) who had atypical chest pain the previous day, but did not develop pain during the test. It is of note that the immediate tracing in S2 and V5 showed only junctional ST depression.

An example of grade 1 horizontal segment depression is shown in Fig. 11 from a 56-year-old man with a 2-year history of angina. The resting tracing showed an inverted T in aVF and flattened T in V6. V5 immediately after effort shows ischaemia but at 2 minutes the T wave has become inverted in V5, V6 and S2 with minimal ST depression. Pain occurred during the test.

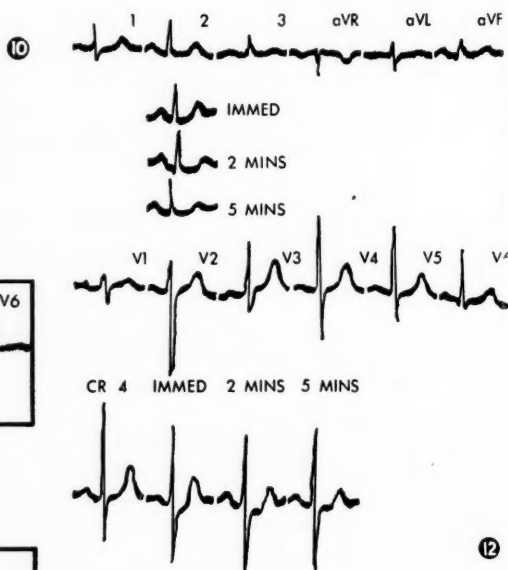


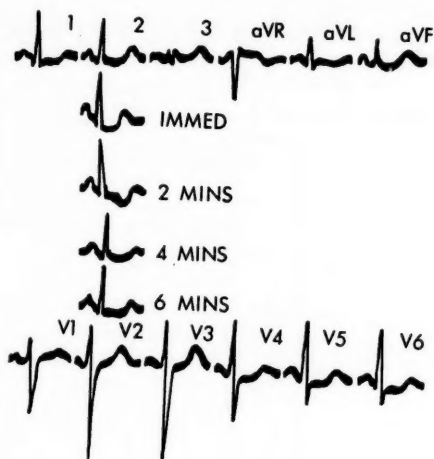
Fig. 12 illustrates grade 2 ischaemia in CR4 after initial junctional ST depression. (Moderate effort on the 2-step test produced characteristic angina).

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Grade 3 depression is shown in Fig. 13 from a 31-year-old man who had angina for 3 months 4 years before and has since remained well. He had no pain during a strenuous test. An initial horizontal depression soon became sagging with an inverted T wave in V5.

A similar picture (Fig. 14) is shown in a symptom-free 63-year-old man with a flat T wave in V6 at rest. No pain developed during the test. The immediate tracing in V4 shows marked sinus arrhythmia—a condition we have seen in several positive reactors but which receives no mention in the literature. We have not seen this arrhythmia in negative reactors.

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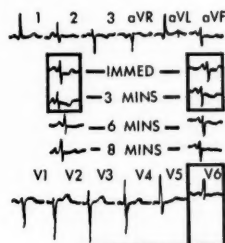
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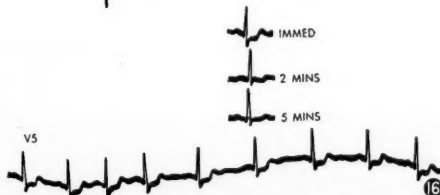
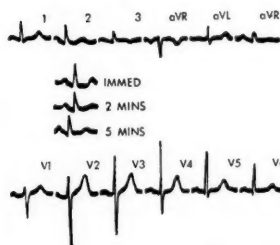
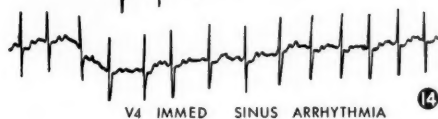
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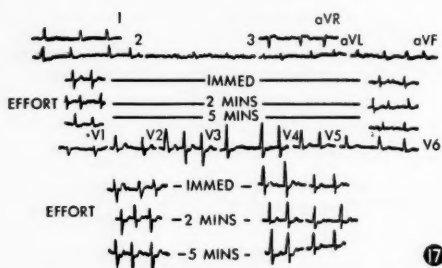
Figs. 15 and 16 also show sinus arrhythmia with ischaemia in positive reactors.

Different leads may show up the ischaemia and this is a good reason for repeating as many leads as is possible with economy of

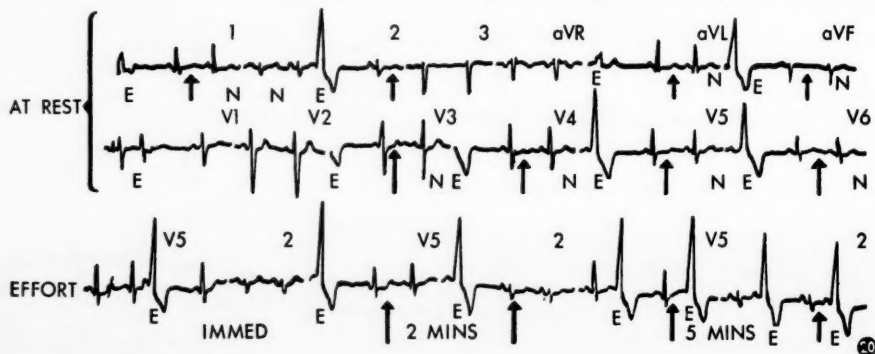
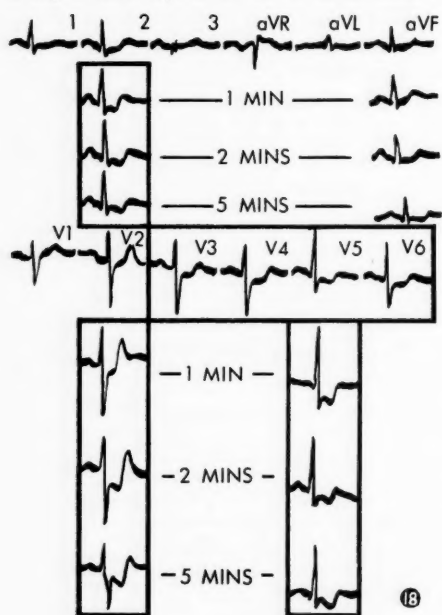


GRADE 3 CHANGES

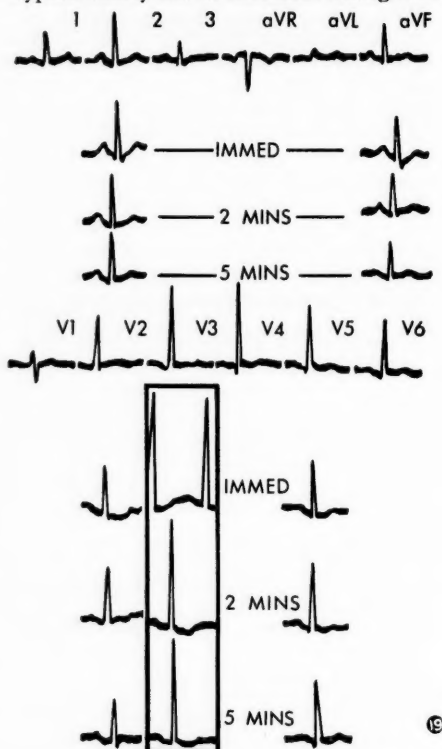




time and effort. Fig. 17 shows that a case of atrial fibrillation of unknown origin was ischaemic in a 54-year-old man, as shown by an effort test which revealed the maximal changes in V2. This also indicates one of the indications for performance of an effort test, i.e. unexplained arrhythmia. After effort there is



a deep S in lead 2, indicating the normal tendency to right axis deviation. Changes maximal in V2 but also present in S2 and V5 are seen (Fig. 18) in a 42-year-old patient with an atypical history but an ischaemic resting ECG.

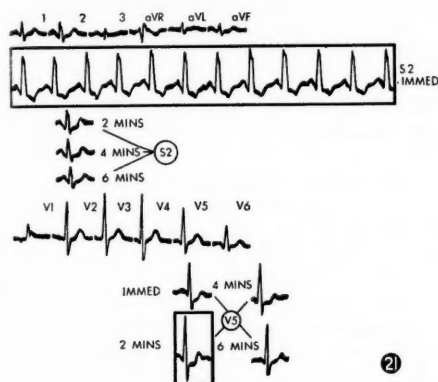


Occasionally, as in Fig. 19, the changes may only be definite in V3. The need for multiple precordial leads after effort thus becomes obvious.

Arrhythmias after effort (apart from sinus arrhythmia, as already mentioned) have a possible but doubtful significance. A curious effect (Fig. 20) is seen in a 57-year-old man with

right supramammary pain on effort. At rest numerous ventricular ectopic beats were present and in each sinus beat following the ectopic beat the T wave was flattened or inverted, especially in V3, V4, V5 as compared to the normal beat (labelled N). Mild effort produced the pain and the ischaemia only showed up in each sinus beat after the ectopic beat, seen best in V5 and S2 at 2 minutes. At 5 minutes he developed bigeminy. Three weeks after the test he sustained a myocardial infarction.

Occasionally effort precipitates an arrhythmia or conduction defect. A man with angina and a normal resting ECG, except for incomplete right bundle branch block shown in V1, developed a complete bundle branch block in S2 in the immediate tracing and typical grade 3 ischaemia in V5 at 2 minutes (Fig. 21).



A symptom-free man aged 49 with a normal resting ECG developed a nodal tachycardia at 2 minutes (Fig. 22). Pain was absent, but he developed ischaemic S-T depression in V5, aVF and S2 with only junctional changes in the other leads. At 15 minutes, when sinus rhythm had been restored, he still had ischaemia in V4.

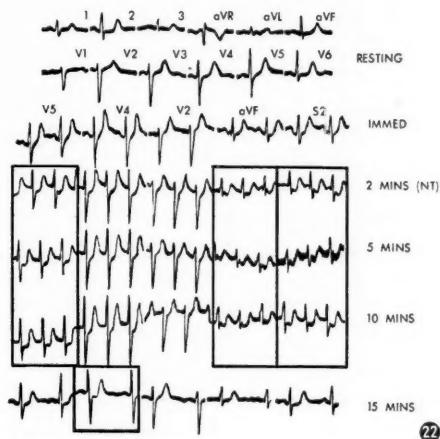
A 55-year-old man with an atypical angina and an abnormal resting ECG showing an inverted T in 2, 3, aVF with a suspicious Q in aVF and flattened T in V6 (Fig. 23) developed multifocal ectopic beats in the immediate tracing and ischaemic sagging in S2 and aVF at 2 minutes, despite the absence of pain. He subsequently developed a myocardial infarction.

It is probable that multiple unifocal ectopic beats have no special significance,⁷ but that multifocal ventricular beats, ventricular tachycardia and QRS conduction defects indicate

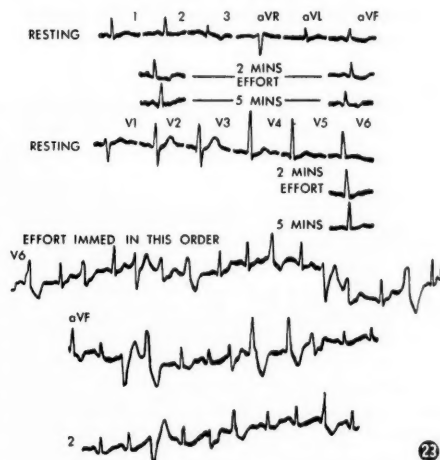
coronary insufficiency. A prolonged P-R interval is of doubtful significance.

Inversion of the U wave is a late, positive finding but is never the sole abnormality.^{4,7}

Table 2 summarizes the criteria for interpretation of the results of an effort test and shows that the results may be broken up into 3 categories—positive, negative, and doubtful.



It is of interest that if true anginal pain is produced by the effort, the test is always positive; but frequently in the absence of pain during the test, ischaemic changes may occur



and thus the test is useful in uncovering latent coronary artery disease.⁴

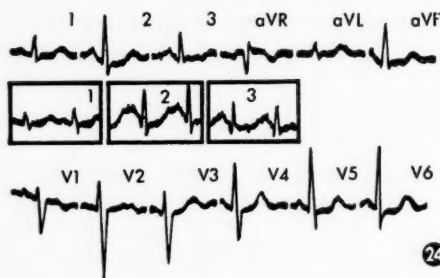
If the heart rate after effort is at least 90 beats a minute, indicating adequate effort, 95%

(or more) of patients with angina and normal resting ECG's develop diagnostic ECG changes.⁴

TABLE 2: INTREPRETATION OF EFFORT E.C.G.

- A. *Evidence of Coronary Insufficiency (Positive Test).*
 1. Ischaemic ST segment depression (Wood).
 2. Probably inverted U waves.
- B. *No Evidence of Coronary Insufficiency (Negative Test)*
 1. No significant change.
 2. Transitory ST junction depression.
 3. Isolated T wave change.
- C. *Changes of Doubtful or Unknown Significance.*
 1. Prolonged ST junction depression with some flattening of ST segment.
 2. Arrhythmias, Ectopics.
 3. Conduction abnormalities.

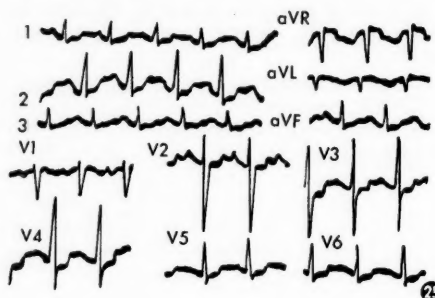
False positive tests are sometimes said to occur,¹ but this must remain an assumption until we can prove that these 'so-called normals' are not, in fact, suffering from latent coronary artery disease. Hyperventilation does not cause true ischaemic depression of S-T segments after effort, although it may lower or invert T waves.^{1, 7, 16} Fig. 24 shows an apparent S-T depression in a woman who hyperventi-



lated to the point of tetany, but careful inspection reveals that there is no true S-T depression. It is our impression, however, that in rare cases hyperventilation may produce almost flat S-T depression in the resting graph, but there is no real change on effort. Fig. 25 is from a young woman with an anxiety state. It shows S-T depression in V2 to V6. Subsequent ECG's and effort ECG's were normal and at no time did she have any cardiac symptoms.

It is indeed probable that a negative response to an adequate exercise test excludes, for practical purposes, latent coronary insufficiency and

clinically significant narrowing of the coronary arteries.^{3, 15} In 251 negative reactors followed for 4 years only 2% (5 cases) had coronary attacks, i.e. the test when negative is 98% certain.¹⁷ While these facts are very significant clinically as a guide, we must remember that statistically, rare individual cases may have a negative test and yet sustain an occlusion.



The dangers of the test are negligible provided that a physician is present and ready to stop the effort at any undue signs of distress. The test should never be left to a technician, as rare cases of myocardial infarction and sudden death have been reported.¹² The test should not be carried out if the patient objects, or the resting ECG shows obvious ischaemic heart disease or generally when the patient is over 60 years of age.

It would thus appear that a properly performed and interpreted effort ECG test is of definite help in the prognosis and diagnosis of coronary artery disease.

SUMMARY

1. The indications for performing an ECG after effort are outlined.
2. The detailed interpretation is discussed.
3. Statistics showing the accuracy of the test are reviewed.

We would like to thank Dr. G. Robb, of the Metropolitan Life Insurance Company of New York, for giving us permission to use several graphs and Tables (Figs. 2, 3, 4; Table 1) and Dr. L. Jacobson, of Bulawayo, for Fig. 25.

We would also like to acknowledge our indebtedness to the Photographic Unit of the Department of Medicine of the University of the Witwatersrand for the photographs of the tracings.

The publication of this paper was made possible by a grant-in-aid from the Swiss-South African Reinsurance Co. Ltd.

REFERENCES

- Graybiel, A. and Allebach, N. W. (1959): Amer. J. Cardiol., **3**, 430.
- Robb, G. P., Marks, H. H. and Mattingly, T. W. (1957): Trans. Assoc. Life Insur. Med. Dir. Amer., **40**, 52.
- Cosby, R. S. and Mayo, M. (1959): Amer. J. Cardiol., **3**, 444.
- Wood, P., McGregor, M., Magidson, O. and Whitaker, W. (1950): Brit. Heart J., **12**, 363.
- Master, A. M., Friedman, R. and Dack, S. (1942): Amer. Heart J., **24**, 777.
- Kossman, C. E. (1958): Trans. Assoc. Life Insur. Med. Dir. Amer., **41**, 101.
- Lepeschkin, E. and Surawicz, B. (1958): New Eng. J. Med., **258**, 511.
- Lepeschkin, E. (1951): *Modern Electrocardiography*, Para. 474-491. Baltimore: Williams and Wilkins.
- Twiss, A. and Sokolow, M. (1942): Amer. Heart J., **23**, 498.
- Mazer, M. and Reisinger, J. A. (1944): Ann. Int. Med., **21**, 645.
- Myers, G. B. and Talmers, F. N. (1955): Ann. Int. Med., **43**, 361.
- Grossman, L. A. and Grossman, M. (1958): J. Amer. Med. Assoc., **179**, 1955.
- Simonson, E. and Keys, A. (1956): Amer. Heart J., **52**, 83.
- Goldberger, E. (1953): *Unipolar Lead Electrocardiography and Vectorcardiography*, 3rd ed., pp. 342-344. London: Henry Kimpton.
- Robb, G. P. and Marks, H. H. (1960): Proc. Soc. Exper. Biol. Med., N.Y., **103**, 450.
- Robb, G. P. (1960): Personal Communication.
- Master, A. M. and Rosenfeld, I. (1959): Trans. Assoc. Life Insur. Med. Dir. Amer., **43**, 70.

THE TREATMENT OF OBESITY IN CHILDREN WITH STELADEX

A COMBINATION OF DEXEDRINE AND STELAZINE

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Almost all children who are brought by parents for the treatment of obesity do not suffer from any endocrine disease. Whatever the etiology, experience has shown that restriction of the intake of food is sufficient to alter the balance in the calorie needs of the body and so will produce a loss of weight. Apart from the cosmetic point of view, no child or adolescent should be allowed, if possible, to continue into adult life carrying a burden of several stones of excess weight.

The treatment is always difficult. To achieve the patients' co-operation is probably the corner-stone in the therapeutic regime. In a few cases an actual endocrine disorder may require treatment but, as a rule, strict adherence to a reducing diet is essentially the treatment of obesity. Attempts to correct wrong eating habits should always be made and both parents and children must be instructed on how best they themselves can contribute to maintaining a regime which is irksome and a burden to all concerned. Drugs like dextroamphetamine sulphate (Dexedrine) do inhibit appetite and as an aid for the child who over-eats, the judicious use of such drugs is most certainly indicated. Like adults, children may find appetite-inhibiting drugs of great value but a disadvantage of this form of treatment may be sleeplessness, irritability and excitability which often mitigate

against the success of a therapeutic regime based on a reducing diet and Dexedrine.

To overcome these complications associated with appetite-inhibiting drugs, amylorbarbitone has often been added to the regime, not always with success with children. The fat child may already be lethargic and amylorbarbitone may increase this in spite of the Dexedrine already prescribed. The advantages of combining a drug like Dexedrine with a tranquillizing drug were considered in the hope that the latter might produce a state of mind whereby a child would not resent the restriction on food and would co-operate better in adhering to the diet prescribed. Such a combination, Steladex, was made available in tablet form for clinical trial some time ago. This combination contains 5 mg. Dexedrine, and 1 mg. Trifluoperazine ('Stelazine').

A double-blind trial was designed whereby patients were selected on a random allocation basis for treatment with either Steladex or Dexedrine alone. The choice of patients was determined solely on the basis that they were over-weight and that weight reduction was essential. Throughout the trial the psychological response of the patients was noted at regular intervals, although they, of course, did not know which tablets they were taking. The period of treatment lasted for 6 weeks in

almost each case and the patients were weighed at regular intervals throughout the course of treatment.

Ten children were treated with Dexedrine and 10 with Steladex tablets. In both groups treated the ages varied from 5 to 13½ years. In the Dexedrine-treated group the weight loss varied from 1½ to 13 lb. with an average loss for the group of 7.6 lb. In the Steladex-treated group the weight loss varied from 2½ to 11½ lb. with an average loss of 5.8 lb. The loss in weight for each group was statistically significant but the difference between the two means was not statistically significant.

These results show, as could have been anticipated, that in promoting loss of weight it is the restriction in the intake of food which is the determining factor. There is no doubt that Dexedrine does inhibit the appetite and that the loss of weight in these 2 groups of patients can only be ascribed to a combination of appetite inhibition and a low food intake. However, it was only by further questioning that other factors were elicited. Those children taking Steladex were apparently much less difficult to control and sleeplessness, irritability and excitability, which were seen in the Dexedrine group, were not seen to the same extent in the Steladex group. One gained the impression that the Steladex combination for this reason is superior to Dexedrine given alone. A state of obesity is certainly associated with psychological disturbances and it became clear that the addition of Stelazine influenced the state of mind of the children taking the drug and there was less resentment and more co-operation in adhering to the diet.

In the treatment of obesity, particularly in children, it is essential, if the treatment is to succeed, that the young patient should not only be made aware of the reason for his dietary restriction but also that he should be persuaded to accept this restriction with good grace. In the older children the derision and jeers of

fellow students is usually an incentive to make them persevere. Even if this is not so, this perseverance may often be aided by the careful use of a tranquillizer which, without side effects, creates a change in the mental attitude and is a good psychological prop in what is certainly a difficult situation.

There is no need to persist in this form of treatment after a satisfactory weight loss has been achieved, because the purpose of treatment is to reduce the weight of the child to a reasonable level and to persuade him to acquire new eating habits to which he must adhere for years to come. A great number of fat children lose their excess weight and achieve normal proportions at the age of 15 or 16 years without any treatment whatsoever, so that drug treatment can be used as a preliminary measure, to be stopped when the child reaches the age when Nature 'takes over'. In later years new eating habits and perseverance will achieve excellent results and drug therapy can be used at periodic intervals when the inevitable relapses occur.

The group of children in this trial has shown that with the dosage used, side effects due to the combination Dexedrine and Stelazine are not seen. Steladex is now manufactured in a Spansule Capsule form so that one Spansule Capsule may be sufficient to maintain control for a 24-hour period.

One can conclude that in the treatment of childhood obesity the intelligent use of appetite-inhibiting drugs combined with a tranquillizer, enables a child to face the restriction in his food with more equanimity and makes threats or exhortations unnecessary.

SUMMARY

The use of Steladex, a combination of dextro-amphetamine sulphate (Dexedrine) and Trifluoperazine (Stelazine), as an adjunct to dietary control, has been shown to be of distinct value in treating obesity in children.

NOTES AND NEWS : BERIGTE

Dr. Edward Epstein, of Johannesburg, recently left for Europe on a 2-months' visit overseas.

He has been invited to lecture at the International Course of Ophthalmology to be held in Barcelona and to take part in the *Symposium on Cataract Surgery*.

Dr. C. J. du Toit, M.B., Ch.B., D.T.M. & H., M.Med. (L. et O.), Ear, Nose and Throat Surgeon, has joined Drs. H. C. Wykerd and D. J. Roux in partnership at Southern Life Building, 101 St. George's Street, Cape Town. (Telephones:— Rooms: 2-1938; Residence: 44-7261).

Mr. H. Berzen, M.Ch.Orth. (Liverpool), F.R.C.S. (Edin.), has joined Mr. W. T. Ross in partnership in orthopaedic practice at 16 Lister Building, Jeppe Street, Johannesburg. (Telephones:— 23-2467; 23-8687) and at 205 Admiral's Court, 31 Tyrwhitt Avenue, Rosebank, Johannesburg. (Telephones:— Rooms: 42-3187; Residence: 44-7149).

INTERNATIONAL CANCER CONGRESS: MOSCOW

The Eighth International Cancer Congress will take place during 22-28 July 1962 in Moscow, under the auspices of the International Union Against Cancer.

The Congress will meet at the Moscow State University (the new building on Lenin Hills).

The National Organizing Committee, established through the initiative of the Ministry of Health of the U.S.S.R. and the Academy of Medical Sciences of the U.S.S.R., is to do all the preparatory work for the Congress.

The official languages of the Congress will be English, Russian and French.

SCIENTIFIC PROGRAMME

The work of the Congress will consist of:

1. Lectures by invited speakers.
2. Panel discussions on selected topics.
3. Section meetings, where proffered papers will be presented.

TOPICS SELECTED FOR LECTURES

1. Role of viruses in the origin of cancer.
2. Biochemistry of cancer.
3. Tumour biology.
4. Experimental research and clinical oncology.
5. New methods of cancer therapy.
6. Radical cancer surgery.
7. New methods in radiotherapy.
8. Cancer control.

The National Organizing Committee is now corresponding with a number of scientists in different countries, recommended by the International Union Against Cancer, with the aim of inviting them to conduct lectures and panel discussions.

PANEL DISCUSSIONS

It is proposed to have 15 panel discussions on the following topics:

1. Viruses in oncology.
2. Immunology and genetics of tumours.
3. Biochemistry of cancer and carcinogenesis.
4. Biology of tumour cell.
5. Epidemiological studies on cancer.
6. Tumour-host relationships and hormone status.
7. Occupational cancer.
8. Aetiology and pathogenesis of liver cancer.
9. Precancerous lesions.
10. Cancer detection.
11. Biological approach to cancer surgery.
12. Supravoltage and high-energy radiation therapy.
13. Chemotherapy.
14. Care of patients with advanced cancer.
15. Cancer education.

SECTION MEETINGS

Besides panel discussions on selected topics, in which the invited speakers will participate, there will also be section meetings with papers limited to 10 minutes.

The following topics have been selected for section meetings:

1. Role of viruses in the origin of cancer.
2. Tumour immunology.
3. Biochemistry of cancer.
4. Biology of the cancer cell.
5. Carcinogenesis.
6. Application of research to clinical oncology.
7. Tumour-host relationships.
8. Precancerous lesions.
9. Radiobiology.
10. Geographical pathology.
11. Carcinoma of the stomach.
12. Carcinoma of the lung.

13. Carcinoma of the ovaries.
14. Carcinoma *in situ* of the cervix.
15. New methods of cancer therapy.
16. Radiotherapy.
17. Long-term results in therapy of cancer.
18. Care of patients with advanced cancer.
19. Cancer control.
20. Miscellaneous.

ENROLMENT

Those entitled to be enrolled as members of the Congress must belong to some recognized scientific or medical organization, or be recommended by such a body. Application for enrolment must be made on Form 1 (which can be obtained from the National Organizing Committee). The deadline for such registration is 1 April 1962.

Members of families may be enrolled as associates. The registration fee (30 U.S. dollars for a member and 15 U.S. dollars for an associate) should be sent before 1 April 1962, to the Vneshtorgbank of the U.S.S.R., account N 0500104. After 1 April 1962, the fee will be 40 U.S. dollars. If cancellations are received before 1 June 1962, the fee will be refunded, less 20%.

APPLICATIONS FOR THE READING OF PAPERS

The National Organizing Committee will accept applications to read papers at section meetings. The reports must concern problems included in the scientific programme of the Congress.

Members wishing to read a paper should fill in and send Form 2 to the National Organizing Committee, also enclosing 3 copies of the abstract of the paper in one of the official languages of the Congress (Russian, English or French). It is desirable that translations into 2 other languages should also be submitted. The text of the abstract should not exceed 250 words. Both the application for a report and the abstract must reach the National Organizing Committee not later than 1 November 1961. Papers received later than this date will be given no consideration. The National Organizing Committee will decide which papers are to be accepted and will, if necessary, consult national cancer organizations of the country of the applicant. The decision of the National Organizing Committee will be forwarded to the authors.

The papers may be illustrated by lantern slides or films, the choice of which must be indicated by the applicant in Form 2.

EXHIBITIONS AND FILMS

There will be an exhibition in the University building during the Congress.

Films devoted to various aspects of study and cure of malignant tumours and cancer control will be shown in a special room.

Members of the Congress wishing to offer films should complete Form 3, giving full details of size of film and showing time, with brief account of its contents.

LADIES' PROGRAMME

A special programme, including sightseeing tours of Moscow and vicinity, will be arranged for the ladies and other associates.

ACCOMMODATION

All foreign members of the Congress will be serviced by Intourists. Preliminary data on Intourist facilities are given below:

1. *De Luxe*: 35 U.S. dollars per person per day: de Luxe accommodation with private bath, 3 meals a day de Luxe menu; 2-3 hours of daily excursions with the use of private car and the assistance of a guide-interpreter, transfers and handling of luggage to and from railway station or airport by private car. De Luxe travellers, desiring to share their room and other accommodation with another person, may pay 25 U.S. dollars per person per day.

2. *1st Class*: 8 U.S. dollars per person per day. Accommodations include: single room with bath for one person, breakfast, transfers and handling of luggage by car to and from station or airport; motor coach from hotel to Congress meetings.

3. *1st Class*: 6.5 U.S. dollars per person per day. Accommodations include: room with bath for 2 persons; breakfast, transfers and handling of luggage by car to and from station or airport; motor coach from hotel to Congress meetings.

4. *2nd Class*: 5.5 U.S. dollars per person per day. Room for 2-3 persons without bath; breakfast, transfers and handling of luggage by car to and from station or airport; motor coach from hotel to Congress meetings.

5. *Accommodation in Students' Hostel*: 4 U.S. dollars per person per day. This includes bed in the University hostel, breakfast in students' dining-hall, transfers and handling of luggage by car to and from station or airport.

For those who wish to arrive by private car, Intourist offers the following facilities:

Single room with bath, 7.5 U.S. dollars per person per day, or a room for 2 persons with bath, 4.5 U.S. dollars per person per day, prices including breakfast, luggage handling and parking.

All prices mentioned above are tentative and subject to slight change.

All formalities (including visas) and payment of charges are to be transacted through travelling agencies acting for Intourist.

Those arriving from countries having no diplomatic relations with the U.S.S.R., or where there are no agents of Intourist, are recommended to apply either to the U.S.S.R. Embassies or to travelling agencies of other countries through which Congress members will pass.

POST-CONGRESS TOURS IN THE U.S.S.R.

Intourist offers post-congress tours to various cities in the U.S.S.R., including Leningrad, Kiev, Sukhumi and Tbilisi. Intourist also offers members and associates visits to resorts on the Black Sea Coast in the Crimea (Yalta) and the Caucasus (Sochi).

All information on such trips may be obtained from Intourist agencies.

PUBLICATION OF PROCEEDINGS

The *Proceedings* of the Congress will be published in Russian in a separate volume by Medgiz (U.S.S.R.). English or French text of *Proceedings* will be published, as usual, by the ACTA U.I.C.C.

Subscriptions for copies of the *Proceedings* will be independent of, and additional to, the registration fee.

CORRESPONDENCE

All correspondence concerning the Congress should be addressed to the National Organizing Committee of the Eighth International Cancer Congress: General Secretary of the Soviet National Organizing Committee, Prof. L. Shabad or Assistant General Secretary, Dr. N. Perevodchikova, Academy of Medical Sciences of the U.S.S.R., 14, Solyanka, Moscow, U.S.S.R.

Soviet National Organizing Committee of the VIII International Cancer Congress.

THIRD WORLD CONGRESS ON THE PREVENTION OF OCCUPATIONAL RISKS

This Congress has been recognized by the French National Safety Institute, in collaboration with the Committee on the Prevention of Occupational Risks of the International Social Security Association and in co-operation with the International Labour Office.

The Congress is being held under the patronage of the French Government and the diplomatic representatives in Paris of 37 countries have also agreed to be patrons of the meeting.

The programme will extend from 22-27 May 1961.

Further information can be obtained from the Secretary General, Third World Congress on the Prevention of Occupational Risks, 9 Avenue Montaigne, Paris 8^e, France.

RULES GOVERNING ANAESTHETIC REGISTRARS PRIZE

Dr. R. A. Moore Dyke has offered a prize for the best paper presented by a Registrar in Anaesthesia at the 43rd South African Medical Congress. The prize will consist of an illuminated scroll and books, to the value of £25.

Entrants must comply with the following rules:

1. They must be full-time members of an Anaesthetic Department in a South African hospital, and the holder of a Registrar or Clinical Assistant post in that hospital, and shall not be registered as a Specialist in Anaesthesia with the S.A. Medical Council.

2. The paper presented must be concerned with original research carried out by the applicant, and be limited to $\frac{1}{2}$ hour with a further $\frac{1}{4}$ hour discussion period.

3. Intention of competing for this prize must be notified to the Secretary of the Scientific Section for Anaesthesia (35 Wale Street, Cape Town), by 30 June 1961, and the completed paper must be in the Secretary's hands by 31 July 1961.

4. The prize will be awarded on the decision of a panel of judges appointed by the Chairman of the Anaesthetic section, and if the number of entries warrants it, this panel will select only those papers which it considers to be of the highest standard for presentation at the Congress.

5. Three factors will be taken into account in awarding the prize: the subject matter and format of the paper, the manner of its presentation before a meeting, and the ability of the author to handle a discussion period on his paper by the meeting.

6. All entries must be accompanied by a letter from the Head of the Anaesthetic Department concerned certifying the status of the entrant.

MEDICAL GRADUATES ASSOCIATION: JOHANNESBURG

DIARY OF EVENTS: MAY 1961

All doctors are cordially invited to attend all Meetings and Ward Rounds.

22-26 May 1961 Annual Students Conference on *Blood Diseases* at the Medical School, at 8 p.m.

25 May 1961 Rand Medical Discussion Club (Medical Graduates Association) together with the Students Annual Conference, at the Harveian Lecture Theatre, Medical School at 8 p.m.
Current Views on Leukaemia, by Dr. H. B. W. Grieg.
Therapy of Acute Leukaemia, by Dr. S. Javett.
Chemotherapy of Chronic Leukaemia, by Dr. D. Durbach.
Radiotherapy of Chronic Leukaemia, by Dr. N. de Moor.

16 May 1961 Medical Association of S.A. (S. Tvl. Branch), at Medical House, 5 Essen St., at 8.15 p.m.
Abortion and Infanticide, by Prof. Jack Friedman.

Clinical Ward Rounds

Mondays General Hospital. Ward 14. Medical. 9 a.m.
Tuesdays General Hospital. Ward 14. Medical. 9 a.m.
Tuesdays General Hospital. Ward 22. Surgical. 8 a.m.
Tuesdays General Hospital. Ward 22. Surgical. 8.30 a.m.
Tuesdays General Hospital. Ward 12. Medical. 10.30 a.m.
Wednesdays General Hospital. Ward 3-4. Professorial Staff Round. 9 a.m.
Wednesdays General Hospital. Ward 14-15. Medical. 9 a.m.
Thursdays General Hospital. Ward 22. Surgical. 8 a.m.
Thursdays General Hospital. Ward 5. Medical. 10 a.m.
Fridays General Hospital. Ward 28. Psychiatric. 9.15 a.m.
Fridays General Hospital. Ward 14-15. Medical. 9 a.m.
Thursdays Fever Hospital. 8.30 a.m.
Fridays Edenvale Hospital. Ward 7, European or Ward B, non-European. 12 noon.
Fridays Coronation Hospital. 2 p.m.
Saturdays Princess Nursing Home. 2nd Floor. Neurology. 9 a.m.
Tuesdays Florence Nightingale. 2nd Floor. Thoracic Unit. 8-9.30 a.m.
Saturdays Florence Nightingale. 2nd Floor. Thoracic Unit. 9.30-10.30 a.m.
Tuesdays Baragwanath Hospital. Lecture Theatre. Surgery. 1.30-2.45 p.m.
Tuesdays Baragwanath Hospital. Ward 17. Paediatrics. 4-5 p.m.

Thursdays Baragwanath Hospital. Lecture Theatre. Cardiology. 2-3 p.m.
Fridays Baragwanath Hospital. Lecture Theatre. Medicine. 2-3 p.m.

Fortnightly Meetings

Tara Hospital, Lecture Room, at 9 a.m.

Wednesdays

10 May 1961 *Hospital Staff*, by Dr. Bersen. Recreation Hall.
 24 May 1961 *Case Presentation*, by Orange Firm. Lecture Hall.
 18 May 1961 Rehabilitation Association for Injured Workmen, Brenthurst Clinic, Room 19, 3rd Floor, at 5.15 p.m.

Weekly Meetings

Institute for the Study of Man in Africa.

8 May 1961 *The Meaning of Race*, by Prof. P. V. Tobias. Harveian Lecture Theatre, at 8 p.m.
 17 May 1961 *Divorce Without Disorganization: The Social and Economic Life of the North Cewa*, by Prof. Max Marwick. Harveian Lecture Theatre, at 8 p.m.
 24 May 1961 *Life of the Egyptian Peasant*, by Dr. H. B. S. Cooke. Chemistry Lecture Theatre, University, at 8 p.m.

S.A. Institute for Medical Research: Lecture Theatre at 5.15 p.m.

8 May 1961 *Laboratory Aspects of an Influenza Vaccination Study on Gold Mines*, by Miss Westwood.
 15 May 1961 *Radio-Isotope Surface Counting Techniques in Haematologic Diagnosis*, by Dr. S. Krawitz.
 22 May 1961 *Paroxysmal Nocturnal Haemoglobinuria*, by Dr. S. M. Lewis.
 29 May 1961 *Statistics as a Tool in Medical Research*, by Dr. H. S. Sichel.

Department of Surgery: Surgical Forum, Harveian Lecture Theatre, Medical School, at 5.15 p.m.

9 May 1961 *Mesothelioma of the Pleura*, by Mr. P. Marchand.
 16 May 1961 *The Surgeon and Hypertension*, by Dr. P. Menof.
 23 May 1961 *Discussion on the Management of the Duodenal Stump*, introduced by Mr. W. H. D. Trubshaw.
 30 May 1961 *Discussion on the Surgical Management of Diverticulitis*, introduced by Mr. J. Wolfowitz.
Saturdays. Hospital Lecture Theatre. Vascular Clinic at 8 a.m.
Saturdays. Hospital Lecture Theatre. Surgical Conference at 9.15 a.m.
Saturdays. Ward 10. Urology Staff Conference at 10.15 a.m.

PREPARATIONS AND APPLIANCES

NAVIDREX* TABLETS

Ciba (Pty.) Ltd. announce the introduction of *Navidrex* (cyclopenthiadiazide) a benzothiadiazine derivative but *not* just another oral diuretic*. Milligram for milligram it is the most potent and most economical sali-diuretic that has ever been offered to the medical profession.

Description: Each tablet contains 0.5 mg. of 3-cyclopentyl-methyl-6-chloro-7-sulphamyl-3, 4-dihydro-1, 2, 4-benzothiadiazine 1, 1-dioxide.

Indications: *Navidrex* can be employed in all conditions for which sali-diuretics are indicated. It has its own antihypertensive effect and may with advantage be combined with more powerful antihypertensive agents such as *Serpasil**, *Adelphane** and *Ismelin**, which can be used in lower dosage than if employed alone.

Contra-Indications: It should not be used in cases of hepatic pre-coma and coma.

As with other sali-diuretics, *Navidrex* may sometimes precipitate an attack of gout during periods of remission in gouty patients or in patients predisposed to gout.

Advantages: Unmatched potency in microgram doses.

Unrivalled economy in use.

Unexcelled tolerability.

Minimal effect on potassium reserves.

Ideal 12-hourly duration of action.

Simple dosage schedule.

Dosage: To ensure a strong diuretic response one tablet (0.5 mg.) should be given daily.

To any patients suffering from extensive oedema in whom it is desired to achieve massive diuresis, it would certainly be preferable to prescribe 2 tablets (1.0 mg.) daily for the first few days. Once the oedema has been resolved, one tablet (0.5 mg.) daily should prove sufficient.

For chronic maintenance therapy, one tablet (0.5 mg.) every second or third day or, alternatively, half a tablet (0.25 mg.) daily. During prolonged therapy supplementary potassium is advised.

Further information may be obtained from: Ciba (Pty.) Limited, P.O. Box 5383, Johannesburg.

* T.M. Regd.

GLUCAGON, LILLY

FOR THE TREATMENT OF HYPOGLYCAEMIC REACTIONS

Description: *Glucagon* hydrochloride is a crystalline polypeptide extracted from the pancreas. When administered parenterally, *Glucagon* produces an increase in blood glucose concentration by converting hepatic glycogen to glucose. Response is usually seen within five to twenty minutes. *Glucagon* may be given by the intramuscular, subcutaneous or intravenous route.

Indications: *Glucagon* is clinically useful in treating hypoglycemic reactions which may occur with Insulin therapy in the management of diabetes mellitus and in terminating Insulin coma induced for treatment of psychiatric disturbances.

Side Effects: *Glucagon* is remarkably free of side effects, but nausea and vomiting may occur with the administration of large doses. However, nausea and vomiting are sometimes symptomatic of hypoglycemia. There are no known contraindications to the use of *Glucagon* as a hyperglycemic agent.

How Supplied: *Glucagon* (Crystalline) as the Hydrochloride is supplied in the lyophilized form with accompanying diluent.

Two package sizes are available:

1 mg. with 1 c.c. of diluent (single dose).

10 mg. with 10 c.c. of diluent (multiple dose).

Further information may be obtained from any Lilly representative or from Ethical Products (Pty.) Ltd., P.O. Box 727, East London.

MADRIBON SYRUP

Roche Products (Pty.) Limited announce the introduction of *Madrison* Syrup.

Description: The long-acting sulphonamide, *Madrison*, sulpha-dimethoxine, has now become available in syrup form 50 c.c. It is a broad-spectrum antibacterial which has shown no toxicity and practically no side effects. The syrup is very pleasantly flavoured and lends itself to accurate dosage by parents and nurses. Each teaspoonful (5 ml.) contains 0.25 g. of *Madrison* substance. *Madrison* syrup is intended for children and young adolescents.

Indications: The indications are for bacterial infections of any system, specifically where no culture and sensitivity tests can be performed.

Dosage: *Madrison* syrup is so designed that the dosage scheme can easily be memorized and risk of overdosage is thereby reduced. For moderate to severe infections the scheme is as follows:

2 to 7 Years: 2 teaspoonfuls *statim*. 1 teaspoonful 24-hourly as a maintenance dose.

8 to 11 Years: 3 teaspoonfuls *statim*. 1½ teaspoonfuls 24-hourly as a maintenance dose.

12 to 14 Years: 4 teaspoonfuls *statim*. 2 teaspoonfuls 24-hourly as a maintenance dose.

At these doses an adequate blood level of the active antibacterial substance, *Madrison*, is maintained slightly above optimum.

Further information may be obtained from the Scientific Department of Roche Products (Pty.) Limited, P.O. Box 6158, Johannesburg.

* Trade Mark.

LIBRIUM 5*

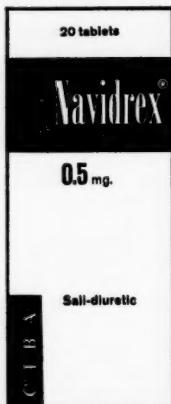
Roche Products (Pty.) Limited announce the introduction of *Librium* 5.

Description: *Librium* 5 is a sugar-coated tablet containing 5 mg. of 7-chloro-2-methylamino-5-phenyl-3H-1, 4 benzodiazepine 4-oxide. *Librium* 5 has been designed as an additional presentation to *Librium* 10 capsules 10 mg., the Roche psycholeptic. The smaller quantity per unit will allow of accurate dosing in paediatrics, geriatrics, cachectic and sensitive patients. The sugar-coated tablet has been designed so that it can even be taken without water.

Indications: *Librium* 5 is a specific anxiolytic with a marked effect on anxiety, tension, unreasoned fear, agitated depression (reactive though not endogenous), autonomic dystonia and migraine. Other specific indications are:

Gynaecology: Premenstrual tension and dysmenorrhoea.

* Trade Mark.



Obstetrics: Pre-delivery anxiety and post-partum depression.

Physical Medicine: As a muscle relaxant when associated with muscle spasms due to anxiety.

Surgery: Pre-operative fear and post-operative discomfort.

Anaesthetics: Premedication adjunct.

Dermatology: Neurodermatitis and itching dermatoses.

Paediatrics: Behaviour disorders, feeding problems.

Geriatrics: Senile agitation, irritability, loss of appetite.

Psychiatry: Idiopathic epilepsy.

Presentation: Sugar-coated tablets in packings of 30's, 100's and 500's.

Further information may be obtained from the Scientific Department of Roche Products (Pty.) Limited, P.O. Box 6158, Johannesburg.

ASTIBAN*

Roche Products (Pty.) Limited announce the introduction of **Astiban**.

Description: **Astiban** is Antimony dimercaptosuccinate sodium salt (TWSb/6), containing 25-26% trivalent antimony. Antimony is bound to the sulphur atoms forming a complex ring structure which has a higher degree of stability than the corresponding oxygen-antimony structures in tartar emetic, catechol and gluconic acid derivatives. The stability of the ring structure containing the antimony is related to the degree of toxicity of this compound. In **Astiban** the antimony is about 30 times less toxic than is tartar emetic, as shown in animal experiments.

Acute Toxicity: The lethal dose of **Astiban** and of tartar emetic was determined in mice. **Astiban** subcutaneously: 90%, 3500 mg. per Kg.; Tartar emetic subcutaneously: 90%, 29 mg. per Kg.

Indications: **Astiban** is to be used in the treatment of *S. haematobium* and *S. mansoni*. It has shown its usefulness in large-scale clinical trials in, firstly, curative treatment and, secondly, suppressive treatment.

Dosage and Administration: **Astiban** should be administered according to the body weight. The total dose in adults lies between 30 and 50 mg. per Kg. with a maximum of 2.5 g. In children under 20 Kg., total doses of 40-60 mg. per Kg. may be given. The higher doses are indicated in *S. mansoni* infestation.

Route of Application: The intramuscular route (a deep injection into the upper outer quadrant of the buttock) is preferred. **Astiban** can, however, be used for intravenous injection, if desired.

Tolerance: Depending on nutritional status, geographical environment, load of schistosomes and other helminths, tolerance can be considered as very satisfactory.

Note: When injections are given on consecutive days more side effects are observed. By extending treatment over 6-9 days, using the alternate day schedule, better tolerance is sometimes obtained without significant decrease in the therapeutic effect.

Side Effects: Anorexia, nausea and vomiting; abdominal discomfort; joint and muscle pains; occasionally activated or latent pyogenic viral or fungal infections; skin rashes, herpes and interdigital pruritus have occasionally been observed. In the proposed dosage, **Astiban** has occasionally produced alterations in T waves but it is not known to affect the cardiovascular, hepatic or renal functions.

Note: The above side effects are mostly benign and transitory and disappear after a short while without after-effects.

* Trade Mark

Contra-Indications: Active tuberculosis, pyogenic infections, febrile conditions, congestive cardiac failure, hepatic insufficiency, jaundice, herpes, massive intestinal helminthic infestations (these should be cleared up first with appropriate anthelmintics before starting **Astiban** treatment. Santonin, however, should not be used.).

Presentation: **Astiban** is presented as a pale yellow dry powder in sterile rubber-capped vials containing 2 g. of the active substance. 20 ml. of pyrogen-free sterile water should be added as a solvent immediately before use. The dissolved substance has limited stability but can be stored in refrigerators and used as long as it remains transparent and practically colourless. Each vial of 2 g. represents the average optimal dose per case.

Distinctive features of **Astiban**:

1. Short duration of treatment.
2. High presumptive cure rate.
3. Satisfactory tolerance.
4. Applicable by the intramuscular route.
5. Suitable for ambulant treatment.
6. Reduction of hospitalization days since daily injections can be given.
7. Indicated for mass treatment and suppressive management of schistosomiasis.

Further information may be obtained from the Scientific Department of Roche Products (Pty.) Limited, P.O. Box 6158, Johannesburg.

DUROMINE

NON-AMPHETAMINE ANORECTIC

'Duromine' presents phenyl-tertiary-butylamine in the now familiar resin bonded ion-exchange form which gives dependable smooth release of the active principle over, normally, a full 12 hours. The widest variations in duration of action lie between 10 and 14 hours from a single capsule, taken in the morning before breakfast.



'Duromine' is non-amphetamine, and in the normal dosage range is non-stimulating; it is specifically designed for the active, alert obese patient who requires no stimulation, or even finds it objectionable.

'Duromine' is available in two strengths, 15 mg. capsules (grey and green) for the person who is of slender build when non-obese, and 30 mg. capsules (grey and maroon) for the patient of heavier build. Both are available in bottles of 15, 30 and 120 capsules.

Riker Laboratories Africa (Pty.) Ltd., P.O. Box 3388, Cape Town.

KARION

South African Druggists introduce Karion—Richter, manufactured by the Lennon Limited Laboratories, under licence to Medimpex of Budapest.

Composition: Karion is 1-piperidino-methyl cyclohexanone-2-hydrochloride.

Mode of Action: Karion in small doses stimulates the chemoreceptors of the carotid body, and in larger doses stimulates both the carotid body and the respiratory centre to cause increased respiration. The increase is in both amplitude and frequency of breathing.

Karion simultaneously induces a slight persistent rise in blood pressure by raising the output of adrenaline from the adrenals, again through both reflex and direct stimulation.

Clinical Experience: Karion is being widely used in maternity homes and clinics, for cases of asphyxia of the newborn, whether this be due to unduly protracted deliveries, drugging of the mothers, or other causes. Karion has been found to be a respiratory stimulant of relatively long action (up to 30 minutes), with no danger of cumulation. In fact, it may be given continuously until regular breathing is restored.

Karion will restore normal breathing in respiratory disturbances due to anaesthetics or electric shock, and in respiratory depression due to morphine, barbiturates, and other forms of poisoning.

Dosage, Toxicity and Side Effects: Karion, for immediate effect, is given by slow intravenous injection. In the infant, half an ampoule (of mite) to start with, is given into the umbilical vein, or into the fontanelle. This may be repeated, or if necessary, a whole ampoule may then be given.

In the adult 1 or 2 ampoules (of forte) are injected intravenously for any respiratory disturbance of central origin.

Treatment may be repeated at intervals of 15-30 minutes.

The margin between effective and toxic doses of Karion is claimed to be greater than those of other preparations used in the treatment of respiratory depression.

Karion does not affect the function of the heart, and has no untoward side effects.

Packing: Karion Mite. Boxes of 5 and 50 ampoules, each containing 15 mg. Karion.

Karion Forte. Boxes of 5 and 50 ampoules, each containing 50 mg. Karion.

Further information may be had from any wholesale branch of the Company and also from: Ethical Promotion Section, South African Druggists Limited, P.O. Box 5644, Johannesburg.

ESKORNADE SPANSULE CAPSULES

Formula:

Isopropamide Iodide 2.5 mg.

Phenylpropanolamine HCl 50.0 mg.

Diphenylpyraline HCl (Histryl) 5 mg.

Eskornade is a unique triple-acting preparation which contains a special drying agent in addition to a decongestant and an antihistamine. Isopropamide iodide, the drying agent, acts to reduce excessive lacrimation and hypersecretion of mucus. The decongestant, phenylpropanolamine, reduces vascular engorgement and often permits blocked sinus cavities to drain. The antihistamine, Histryl, reduces sneezing, rhinorrhea, and also itching of the eyes. Acting together, these three agents combine to provide outstanding relief from upper respiratory distress.

The therapeutic effect of Eskornade begins promptly and, because it is a 'Spansule' sustained release capsule, continues for 10 to 12 hours with a single dose.

Indications: Congestion and hypersecretion in the nasal and paranasal sinuses associated with: the common cold, nasal allergy, acute and chronic rhinitis, influenza and sinusitis.

Contra-Indications and Side Effects: Glaucoma and prostatic hypertrophy. Use cautiously when severe hypertension is present. Side effects are slight and transient. These include dry mouth, drowsiness, insomnia and blurred vision.

Dosage: For adults and children over 12: One Eskornade Spansule capsule every 12 hours.

Presentation: Spansule capsules in containers of 30.

Further information may be obtained from: SKF Laboratories (Pty.) Limited, P.O. Box 38, Isando, Tvl.

REVIEWS OF BOOKS

THORACOTOMY

Techniques of Thoracotomy. By B. T. le Roux, M.B., Ch.B., F.R.C.S.E. (1961. Pp. 91 + Index. With 65 Figs. R 5.50 plus 15c. postage abroad).

Edinburgh and London: E. & S. Livingstone Ltd.

This book is especially welcome, as it deals with an aspect of thoracic surgery usually neglected in textbooks. All acquainted with thoracic surgical procedures know the importance of accurate access. A misplaced incision through the rigid chest wall can make an easy intra-thoracic procedure extremely difficult and, at times, even hazardous.

The text contains detailed descriptions of standard thoracic incisions, such as lateral thoracotomy, thoracotomy, vertical midline sternum-splitting thoracotomy, scapula-displacing incisions, etc. The book is extremely well illustrated, and the text is

made up mainly of descriptions of these pictures. This makes it easy to follow the various steps in the thoracotomy.

There is very little to criticize in this book, except for 2 minor points. Firstly the author deliberately avoids diathermy and, secondly, he advises interrupted suturing of all the layers of the chest wall. Although with modern anaesthesia hurried surgery is unnecessary, it is important not to extend the operation time unnecessarily. As it is, thoracic surgical procedures are time-consuming, and the use of diathermy for haemostasis and continuous suturing of the chest wall are valuable. This applies especially to open heart surgery, where fatigue for the surgeon is to be avoided and it is important to save time.

This reviewer recommends this book to all those who desire to embark on this field of surgery and also, for that matter, to the more senior, busy surgeon, as a reminder of the smaller but important details of access, which make for easier, neater and better surgery.

WILLIS ON TUMOURS

Pathology of Tumours. Third Edition. By R. A. Willis, D.Sc., M.D., F.R.C.P. (1960) Pp. 1002 + Index. With 500 Figs. R10.50 plus R0.30 postage).

London and Durban: Butterworth & Co. (Publishers) Ltd.

The first edition of this work appeared in 1948; it was reprinted in 1948 and 1949. A second edition followed in 1953, and new advances and popular demand have made it necessary for the publication of the third edition within a comparatively short time. Professor Willis requires no introduction. His wide experience, which has been reflected in his many publications, has made him a well-known and authoritative figure.

The plan of the third edition follows that of its predecessors. Part I deals in 202 pages (12 chapters) with the general pathology of neoplasia; Part II comprises 800 pages (50 chapters) and is concerned with the consideration of tumours of organs and sites. Two short *Appendices* deal respectively with conditions easily mistaken histologically for malignant tumours and some lesions associated with malignant tumours. All chapters have been revised and the text has been added to or rewritten on 15 different subjects.

Willis claims that the book is addressed primarily to pathologists, research workers and senior students and as a standard work of reference in the field of tumour pathology has been well received by most reviewers. Yet some statements must be accepted with the greatest reserve, e.g.:

At p. 433:

'Primary carcinoma of the liver is a diagnosis which can be established only by thorough necropsy';

At p. 365:

'Statistical evidence alone now makes it certain that cigarette smoking is an important cause of cancer';

At p. 685:

'The giant-cell tumour of bone arises from and consists of bone formative (as opposed to marrow) cells, which possess the attributes of osteoclasts.

Furthermore, in a volume intended for pathologists and research workers there are some notable omissions. This reviewer searched in vain for some account of the Kaposi sarcoma; haemangio-pericytoma is dismissed in a brief paragraph and the systemic effects associated with malignant tumours are dealt with in a short appendix of one page. One must comment also on the lack of depth in the accounts of individual tumours. Perhaps the day is now past when the pathology of all tumours can be described and discussed fruitfully or even adequately in one volume. Certainly, as a work of reference for the specialist pathologist this book cannot be said to have achieved its aim.

ORTHOPAEDICS

Orthopaedics. By George Perkins, M.C., M.Ch., F.R.C.S. (1961). Pp. 964 + Index. With 577 Figs. R12.60).

London: The Athlone Press.

This volume contains the accumulated experience and practical philosophy of a well-known senior clinical teacher of orthopaedics. Comprehensive references are not included and selected references have been deliberately haphazard.

Each item is covered briefly and dogmatically, tending to mislead the student into thinking that

the opinion expressed is the only tenable one. Arbitrary opinions are expressed on a take-it-or-leave-it basis. Intermittently there is a departure from the personal view in favour of the quoted opinion of some other writer without confirmation or repudiation; as much as to say: 'Believe it if you like.'

Tumours of bone are dealt with briefly. A clear guide to treatment is not always offered, e.g. at what stage cobalt bomb therapy should be followed by amputation is not made clear. Rather vague statements, such as 'pre-amputation radiotherapy is probably worthwhile', will still leave the practitioner in doubt as to whether the implied delay is justifiable.

In secondary malignant diseases hormone control is just mentioned and cytostatic agents are not discussed. On the whole the tumour section is clear, if intentionally over-simplified.

Amputations are dealt with cursorily. The suction socket prosthesis is not mentioned, nor is the Continental preference for suturing muscles over the end of the stump. Hindquarter amputation, a rare operation even in a specialized unit, is given a full page whereas everyday amputations are dealt with in a few lines.

In rheumatoid arthritis the recent orthopaedic studies of the mechanical effects of osteophytes by Vaughan Jackson are not mentioned, nor are the many ingenious surgical procedures for the alleviation of deformity and improvement of function, as described by Boyes, Littler and Riordan. In providing answers to problem cases the book is strangely disappointing, e.g. is it reasonable to arthrodesis both knees for osteoarthritis? The problems of bilateral severe osteoarthritis of the hip, the relative merits of double arthroplasty, displacement osteotomy or arthrodesis or combinations of these, are not clarified, although mono-articular arthrodesis is dealt with adequately. The central dislocation type of hip fusion is ignored. In recurrent dislocation of the shoulder, the muscle splitting atraumatic approach is not mentioned. Instead the coracoid and subscapularis cutting is still advised—surely a needlessly damaging approach to a repair of the labrum.

Bicipital tendonitis and attrition of the tendon is not mentioned as a common factor in frozen shoulder. On the other hand, foot deformities and treatment are dealt with in detail. Interesting radiographs are shown of spondylolisthesis progressing under observation. Operation for prolapsed disc is advised only as a last resort when everything else has failed—surely a disastrous policy. The interlaminar operation is not described. Tuberculosis of bones and joints as well as tuberculosis of the spine and paraplegia are very fully dealt with. The chapter on peripheral nerves is clear and concise.

This book consists of nearly 1,000 pages and contains a great deal of useful basic teaching, especially on the examination of various regions. The type is easy to read. The general layout and the quality of paper are excellent.

The best part of the book is the numerous beautiful illustrations and reproduced radiographs. One finds the volume interesting to browse in. It could be read for relaxation but it is irritating in its inadequacies and cannot be recommended to the orthopaedic postgraduate student. The practising orthopaedic surgeon will not find in it the answers to his problems, as the book appears to cater essentially for the undergraduate who wishes to acquire an elementary knowledge of orthopaedics in a short time.

A POCKET MEDICAL DICTIONARY

Blakiston's Illustrated Pocket Medical Dictionary. Ed. by Normand L. Hoerr, M.D. and Arthur Osol, Ph.D. (1961). Pp. 985. With 60 illustrations. Plain \$5.75. Indexed \$6.50.

New York: McGraw-Hill Book Company, Inc.

This work is based on that massive parent of medical dictionaries, *Blakiston's New Gould Medical Dictionary*. The problem of the editors has been to condense into a smaller compass the material of

importance to medical students and persons in allied professions, including secretaries, assistants, etc.

This very conveniently sized pocket edition runs to close on 1,000 pages and includes 24 plates of illustrations, many in colour.

There is a key to pronunciation and information in the various Tables has been brought up to date, as typified, e.g. by the inclusion of a list of radioactive and other isotopes referred to in medicine and biology.

This pocket medical dictionary should prove a boon to all those who work in close association with medical practitioners.

CORRESPONDENCE

ELECTRO-CONVULSIVE THERAPY

To the Editor: Dr. Feldman's wide clinical experience in the treatment of depressive conditions is well reflected in his paper on physical methods of treatment.¹ As the local protagonist of ECT, his views merit our closest attention.

One of the main arguments in support of the claim that psychiatry has effective methods of treatment rests upon the results claimed for cerebral electroshock. Yet the few controlled studies which have been carried out to date^{2,3} show no statistical superiority of treated over untreated groups. The evidence suggests that ECT is effective only in speeding up remission which has already begun in a depressive illness.

Dr. Feldman ignores the two main disadvantages of ECT, viz:

1. *Brain damage* ranging from small haemorrhages to cerebral disturbance of an epileptoid variety⁴ (which some patients show for as long as 6 months after ECT).

2. *Amnesia* and similar memory defects. (There nearly always is a temporary cognitive loss but

permanent impairment of memory is by no means uncommon.⁵)

It could be argued that shock treatment should be confined to depressive cases in which definite suicidal risks are present. Unfortunately, I have many cases on record where ECT has been applied to phobic and obsessional cases with a resultant exacerbation of generalized and specific anxiety.

REFERENCES

1. Feldman, M. B. (1960): *Med. Proc.*, **6**, 621.
2. Huston, P. E. and Locher, L. M. (1948): *Arch. Neurol. Psychiat.*, **60**, 37.
3. Karagulla, S. (1950): *J. Ment. Sci.*, **96**, 1060.
4. Levy, N. A., Serota, H. M. and Grinker, R. R. (1942): *Arch. Neurol. Psychiat.*, **47**, 1009.
5. Janis, I. L. (1950): *J. Nerv. Ment. Dis.*, **111**, 359.

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DR. M. FELDMAN'S COMMENTS

To the Editor: Thank you for the courtesy of permitting me to comment on the letter *ECT Therapy* addressed to you by Arnold A. Lazarus, M.A., Ph.D.

1. With regard to your correspondent's curious reference to myself as 'the local protagonist of ECT,' it was, it seemed to me, made obvious in my contribution to the local symposium on *Modern Therapy in Depression*¹ that, before considering the *Physical Methods of Treatment*,² *per se*, the distinction had to be made clear between *melancholia* (manic-depressive depression) in which 'shock treatment' might be indicated; and the 'neurotic,' 'hysterical' or 'reactive depressions,' in which psychotherapeutic methods are preferred. Has this attempt at clarification, not only of the indications but also of the *contra-indications* to ECT, been in vain? It was further emphasized what diagnostic/therapeutic confusion and social stigma already stood in the way of the unfortunate melancholic achieving, once the diagnosis was established, the electro-

cerebral treatment which would rapidly and effectively, in the majority of cases where it was indicated, relieve his profound agony of mind. Let not confusion now be worse confounded by the possible attribution of misplaced enthusiasm to the EC therapist!

Modern (and local) psychiatric teaching not only emphasizes the attempt to recognize the *nature and quality of the patient's symptoms*, but also the appropriate handling of the *patient* who has the illness³ and not only of the *illness* which the patient has. This involves evaluation of the total life history of the patient, and of his psychological and physical status, leading to the formulation of a *programme of treatment* which includes psychotherapy, environmental manipulation, the judicious use of the amphetamines (one clinical psychologist at least has learnt of the virtue of this type of drug therapy and communicates to referring medical practitioners news of its value, in some of the depressions),⁴ sedation, the

newer antimelancholic drugs and, where properly indicated, the discerning use of electro-convulsive treatment, so that this latter is, where it is considered necessary, part of a therapeutic plan and not a panacea for all depressive illness.

2. Clinical psychologists who have served the full period of clinical internship at local training hospitals should surely, it was hoped, have achieved awareness of the smaller place of ECT in the larger psycho-biological scheme of things; yet your correspondent goes on to say that 'one of the main arguments in support of the claim that psychiatry has effective methods of treatment rests upon the results claimed for cerebral electroshock.' This is news to me—are the authorities in support of this viewpoint medical, lay or apocryphal?

The daily task of the psychiatrist as is well known involves the care of the organic deliria, the manias, the schizophrenias, the psychoneuroses, psychosomatic and character disorders (including alcoholism and drug addiction), for many of which psychiatry has effective methods of treatment. We do not, however, seek to claim that we 'cure' 90% of our cases—perhaps our techniques are at fault—perhaps our selection of cases for treatment too indiscriminate! For the dementias, many of the schizophrenias and major character disorders, it is true that at present we have not 'effective' therapies but even in these we accept the responsibility of arranging for care, supervision and, where necessary, control.

3. It is then further argued that the 'few controlled studies which have been carried out to date show no statistical superiority of treated over untreated groups. The evidence suggests that ECT is effective only in speeding up remission which has already begun in a depressive illness.'

Of the 2 references quoted in support of this latter argument, the first⁵ states under 'Summary and Conclusions':

'It is concluded that electric shock therapy is indicated in the treatment of manic-depressive psychosis (depressive type, mixed type, perplexed type and type with paranoid features). The basis of this conclusion is that shock therapy produces a rate of recovery as high as that of spontaneous recovery, reduces the incidence of suicide, probably prevents deaths and shortens the duration of depressions.' (Italics inserted), and the second⁶ states: 'Although the statistical evidence in this survey does not prove that ECT increases recovery rates, decreases duration and prevents recurrence in depressive states, clinical observation testifying to its value cannot be disregarded. Convulsion therapy frequently ameliorates symptoms and renders the illness more bearable' (Italics inserted), 'and it would therefore appear to act by stimulating the mechanisms which bring about spontaneous recovery. Increased knowledge regarding its mode of action may therefore lead to increased understanding of depression as a clinical entity. The absence of such information stresses the continued necessity of detailed study of the individual patient and his symptoms in their total setting.' (Italics inserted.)

Further, another reference quoted by your correspondent⁷ states (with regard to ECT) under 'Conclusions':

'Of 23 patients, 14 recovered or improved and 9 did not improve. Early recurrences were included in the category of no improvement. In contrast to previous forms of treatment, the striking feature was the spectacular rapidity of recovery of the patients who were cured.' (Italics inserted.)

4. Your correspondent tells us that 'two main disadvantages of ECT are ignored': the first, 'brain damage ranging from small haemorrhages to cerebral disturbance of an epileptoid variety.'

The reference given⁸ quotes under 'Conclusions': 'Evidence of disturbed cerebral function was present in 50% of the patients as indicated by changes in the electro-encephalogram and in 45% of the patients as shown by changes in intellectual function. In the most severely affected patients epileptoid disturbances in the electro-encephalogram developed, consisting of 3 per second waves, bicuspid and dicrotic waves, spike and wave formations and greatly increased amplitude. The post-therapeutic clinical evidence of impaired cerebral function consisted of an "organic" type of sensorial and intellectual impairment. Recovery from these disturbances of cerebral function occurred in most patients in a few weeks. (Italics inserted). In the more severely affected patients, evidence of impaired cerebral function sometimes lasted as long as 6 months.'

With regard to 'Brain damage' with 'small haemorrhages' alleged by your correspondent to occur after ECT, his reference⁹ indicates:

(a) '4 out of 7' 'monkeys' after 30 to 147 minutes of Metrazol convulsions' (the average patient has a total of half to one dozen treatments of 30 seconds each) showed 'cellular changes and subarachnoid (Italics inserted) haemorrhages.'

(b) '6 psychotic patients' treated with Metrazol convulsions' showed 'marked hypertrophy and hyperplasia of astrocytes and to a lesser degree of microglyocytes' etc.

With regard to ECT however, 'Anatomic studies on animals' by Cerletti and Bini, to whom is owed the original use of electroshock, revealed only reversible changes in the brain (Italics inserted). The authors who (even including Metrazol) go no further than to suggest that 'structural damage' may (Italics inserted) result from convulsive therapy, conclude (after discussing the 'few pathological studies' on ECT) 'our data fail to shed any light on the character of the damage to the brain, but its reversible nature is suggested by the return to normal, or approximately normal function (Italics inserted). Further observation on our patients is necessary before definite conclusions can be drawn regarding the possibly permanent or wholly reversible nature of these disturbances.' What admirable and scientific reserve! Where now the 'Brain damage ranging from small haemorrhages,' etc.?

The second 'main disadvantage of ECT' 'ignored' according to your correspondent is that 'there always is a temporary cognitive loss but permanent impairment of memory is by no means uncommon.' The reference quoted¹⁰ states under 'Summary and Conclusions':

'(1) Definite and consistent evidence of circumscribed (Italics inserted) amnesias was found following electric convulsive treatments. Approximately 4 weeks after the termination of treatments, every one of the 19 electroshock-treated patients was unable to recall some (Italics inserted) of the memories of past experiences which had been elicited in pre-treatment interview. Such failures occurred so infrequently among the 11 control patients as to be almost negligible.

(2) In a follow-up study on 5 of the electroshock-treated patients it was found that in each case most of the instances of retroactive amnesia persisted as of 2½ to 3½ months after termination of the treatments. This finding bears out the preceding one

in supporting the general conclusion that a series of electrically induced convulsions, as administered in standard psychiatric practice, produces *circumscribed* (Italics inserted) amnesias for past experiences which persist beyond the *usual period of recovery during which the temporary organic reactions to the treatments clear up.* (Italics inserted.)

It goes on to say:

(3) In the post-treatment interviews of electro-shock-treated patients it was noted that in addition to the definite retroactive amnesias there were numerous instances of recall failures which were *purely temporary* (Italics inserted) in that the patient was subsequently able to remember some material which he could not recall at first. If it is assumed that such temporary recall failures involve the same basic defect as the more persistent recall failures, the *post-treatment amnesias may be regarded as recoverable rather than as presenting a permanent retention loss.* (Italics inserted), yet your correspondent says (forebodingly) 'permanent impairment of memory is by no means uncommon.'

Furthermore, the author adds:

(4) There is some evidence that following electric convulsive treatments extra effort is required for overcoming the difficulty in producing personal memories, when the patient attempts to remember his past experiences. *Observations on motivational aspects of the post-treatment amnesias provide some support for the hypothesis that those memories which tend to evoke guilt, lowered self-esteem, or other painful affective reactions may be less likely than others to be recalled because the patient is motivated, consciously or unconsciously, to avoid expending the added effort required for producing them. According to this hypothesis, the general difficulty in producing personal memories may facilitate the forgetting of certain (disturbing) past experiences. This might prove to be a basis for explaining the occurrence of sustained amnesic gaps following electro-convulsive treatments,* so that, you see, your correspondent's own reference reveals that what he has considered a 'disadvantage' may (a) not be such (b) could be 'hysterical' (comparisons will be avoided) and (c) in any event not worth fussing about, let alone magnified by implication and association to create alarm and despondency about 'brain damage,' forsooth!

5. Your correspondent then (from the wealth of his own experience and wisdom, it is presumed, as no reference is given), goes on to suggest: 'It could be argued that shock treatment should be confined to depressive cases in which definite suicidal risks are present.'

It is difficult to see how this 'argument' can be used even if other considerations are left aside, regard being had *only* to the quotations culled from *your correspondent's own references.* The assessment of what your correspondent refers to as 'definite suicidal risks' cannot in 'depressive cases' be alone permitted to be an indication for ECT, *without skilled evaluation of the 'depressive case'* along the lines already indicated. For example, the hysterical depressive, whose suicidal attempt is 'designed' to put pressure on the environment, is *not* considered to be a candidate for ECT in the absence of melancholic symptoms. Contrariwise, though every more than mild melancholic is a potential 'definite suicidal risk,' ECT is not indicated for all—they may respond rapidly to other measures. The mild but *prolonged* melancholic, on the other hand, in whom the depth of manic-depressive depression is not sufficiently profound to lead to preoccupation with suicide, *should not be denied the benefits of ECT* if other

methods of treatment fail to give relief *within a reasonable period of time.*

6. Your correspondent concludes by saying:

'Unfortunately I have many cases on record where ECT has been applied to phobic and obsessional cases with a resultant exacerbation of generalized and specific anxiety.'

Now, it is well known that many phobic-obsessionals do have melancholic episodes which necessitate *and benefit from ECT* at the expense of leaving some of them with a (not necessarily new) phobia for the treatment. This has not, however, prevented many of them coming for electrical treatment again when their melancholia has relapsed or recurred (sometimes in spite of psychotherapies applied in the interim). It is also true that melancholic phobias, obsessions and 'anxieties' are not infrequently *resolved by ECT*, so that one must beware of 'exacerbating' the confusion by 'generalization' from the 'specific' cases ('many'! how many?) which filter through to the clinical psychologist whose hospital experience is limited, *even if extended to the maximum of the minimum period required for registration as a medical auxiliary*, so that he is not enabled to follow-up over long periods a great number of cases of melancholic illness, and is therefore likely to fail to see in proper perspective the (possibly retrospectively exaggerated) post-ECT 'anxiety' exacerbation.

7. I hope you will agree with me, Sir, that 'hit-and-run' critics should not be permitted to substitute warmth for wisdom, nor, in their haste to deal with the things they suppose to be wrong, be allowed to upset the things which are right.

8. Allow me in conclusion to apologize for the length of my comment—it looks very much like the proverbial sledgehammer being used to swat the proverbial mosquito—but as indicated in a recent paper, *Drugs in the Treatment of Depression:*⁹

'Patients can nearly always be helped out of an attack of depression, most of them fairly speedily, *if only the correct diagnosis is made and the appropriate treatments are given.*' (Italics inserted) and in view of the fact emphasized by the author, that 'the classical melancholic depressions—those depressions needing and often being so quickly and certainly helped by electric shock treatment' (Italics inserted) are far from always being helped by the new anti-melancholic drugs, one cannot allow ill-informed prejudice to stand in the way of the remarkably effective, though empirical, anti-melancholic electrotherapy.

REFERENCES

1. Med. Proc., 1960, **6**, 626.
2. Med. Proc., 1960, **6**, 621.
3. *Psychiatry in a Nutshell*, S. Afr. Pract., 1960, **7**, 11.
4. Personal communication: A. A. Lazarus.
5. Huston, P. E. and Locher, L. M. (1948): Arch. Neurol. Psychiat., **60**, 37.
6. Karagulla, S. (1950): J. Ment. Sci., **96**, 1060.
7. Levy, N. A., Serota, H. M. and Grinker, R. R. (1942): Arch. Neurol. Psychiat., **47**, 1009.
8. Janis, I. L. (1950): J. Nerv. Ment. Dis., **11**, 359.
9. Sargent, W. (1961): Brit. Med. J., **1**, 225.

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